Improved Communication

—HYGIENIC STANDARDS FOR DAILY INHALATION—

The Donald E. Cummings Memorial Lecture

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EXPERIENCE convinces us that we humans are unique in the universe. Because of our ability to communicate, we may be well on the way toward emerging into a new level of biological existence. Each individual may eventually share completely all past and present experiences of the species. Each may act in concert with his fellows toward common ideals and goals, still retaining his own individuality. Real progress in this direction has been made during the past ten thousand years through developments of recording, duplicating and retrieval techniques. Despite brief back-sliding, there has furthermore been real spiritual progress, and an increase in the proportion of men of good will. The next ten thousand years should bring substantial achievements in communication upon higher levels, perhaps even through inarticulate contact of mind with mind. There are hints that what some have called the world mind may come into being before the present human species evolves physically into whatever new species its body is tending toward.

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However, until the world mind develops, we are forced to depend upon more prosaic means of communication. Not so many generations ago, a natural philosopher like Roger Bacon, whom today we call a scientist, could live a full life investigating the secrets of nature, feeling no need and finding no opportunity to communicate his discoveries and his conclusions to a living soul. He could bury his achievements in code, making them difficult for posterity to unravel. We do not have such people and such situations today. Each one of us benefits from the current division of labor, of experience, and of knowledge. Each one of us is a unique specialist, depending upon a multitude of other unique specialists for the achievement of our aims, for our very existence. If nothing else motivates us, simple self-interest should dictate that each one of us ought to make public all that he has learned, in order that his fellow specialists may use it to help us all.

Communication

A YEAR AGO Sterner (1955) expressed the situation in more concrete terms. We comprise persons separately trained in highly specialized fields, led after training to cooperate in the common aim of providing means by which technological developments in occupation may be utilized in a manner compatible with complete health. He said, "We must provide a fluid and effective means of communication between the chemist, the engineer, the physicist, the toxicolo-

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gist, the physician, and the other specialists brought into industrial hygiene. There must result from this interchange of ideas not only an appreciation of each team member's contribution, but an ability actually to bridge the gap between the disciplines, to synthesize, from the offerings of each of the fields, the solutions to the ever more complicated problems. Each member specialist must not only contribute the information he is most qualified to give, but also must encourage a sympathetic, intelligent, and mutual understanding."

In this matter of mutual understanding, we are very much like the inhabitants of Looking Glass Land. You remember that the White Queen told Alice, "Now *here*, you see, it takes all the running you can do, to keep in the same place. If you want to get somewhere else, you must run at least twice as fast as that." With the entry of new people into our profession and the recognition of new constituent specialties such as health physics and atmospheric pollution control, each with a tendency to keep to itself, are we running fast enough even to stay in the same place? We all feel there are too many conferences and too many committees to leave us time to do our daily work. On the other hand, if we are to do our work as well as it can be done, we must be in constant communication with related specialists, because each one of us fully knows only one facet of his own problems.

Communication is not a simple process. It requires an informed speaker or writer who can express himself at the level of comprehension of his audience. It requires an audience which wishes to receive communication. It must be carried on with words, those abstract symbols for reality, each of which has a different meaning to each individual, shaded by his entire past experience. Only a newly coined word is free from ambiguity, and it remains new for only a brief interval. Most important of all, successful communication requires what is today known as feed-back. By this, the speaker hears his audiences' impressions. He can correct and amplify his words until he thinks his audience truly perceives his meaning. A leisurely conversation can be effective communication through feed-back; an article in a journal is likely to be poor communication because feedback is inadequate.

The relatively new specialty of industrial toxicology has already contributed to the equally new profession of industrial hygiene by means of communication. The toxicologists are doing an acceptable job for those people who recognize their need for toxicological information and opinion. The job could be done better, but it is at least acceptable. Success is by no means as great in helping those people who do not recognize their need for help. Once more communication cannot succeed unless the audience desires to receive communication.

Acceptable Concentrations

THE MOST important communication within industrial hygiene, and between our profession and others, may be the collection of judgments upon acceptable concentrations of contaminants in working atmospheres. During what may be called the age of chaos, every experienced industrial hygienist had a few values uniquely his own, drawn from his own experience. For less familiar substances, he borrowed more or less judiciously from the values cherished by his professional colleagues. Some degree of unanimity was brought about when the United States Public Health Service values, based on its long-time collective experience in industrial hygiene, were published in a manual (USPHS 1943). Further unanimity followed publication of the values collected and extended by Cook (1945). In 1947 the American Conference of Governmental Industrial Hygienists published its first list in the Industrial Hygiene Newsletter (ACGIH 1947). In the next two years (ACGIH 1948, ACGIH 1949) revised lists were privately circulated to the members of the Association. Then (ACGIH 1950) publication took place in a scientific journal, and each year thereafter a revised list of threshold limit values has appeared in the scientific literature, and has been generally accepted.

The contributions of Cook (1945) in unifying opinion, and in weighing threshold limit values then in use, judging new data and proposing a list of 129 values, are worthy of high regard. Among the 238 values for substances other than mineral dusts in the current list (ACGIH 1956) of established and tentative threshold limit values are 54 of those which were first pro-

posed in Cook's list, some as definitely established, others to be used cautiously until verified by actual experience.

Not since Cook has anyone published a summary of the data which serve as bases for the selection of specific threshold limits. The privately circulated documents which give some of these data (ACGIH 1953b, 1954b, 1955b) cannot be considered to be publication, although they are freely available to any person.

Threshold limits are, and must continue to be the products of judgment, important if true. Some few truly represent their definition and are approximations of the maximum concentrations which can be inhaled continuously and repeatedly without injury to health. These may possibly be fit parameters for incorporation into codes and regulations. Many of the threshold limits are well below concentrations which can injure health. They represent current judgment as to concentrations to which, good practice dictates, men may be expected to subject themselves. These do not seem fit parameters for regulations. In a particular operation, if possible, it is desirable to maintain concentrations below the bench mark by reasonable ventilation and precaution. It is always best to reduce exposure to chemicals to the lowest practical level.

Previous Suggestions for Improvement

DURING the Ninth Annual Congress on Industrial Health, the writer was chairman of the Committee on Chemical Agents (1949). The report of the 16-man committee devoted considerable attention to praising the development of threshold limits, and to suggesting ways in which their presentation could be made more useful. Since that report, two developments have taken place along lines desired by the Committee. The annual table of the Threshold Limits Committee of the American Conference of Governmental Industrial Hygienists is now published in the Archives of Industrial Health, removing the earlier implication of quasi-legal status arising from its appearance in the Industrial Hygiene Newsletter of the Division of Industrial Hygiene, U.S. Public Health Service. In 1954, the Committee on Threshold Limits of that Association began to supplement its table of accepted values with a list of tentative values.

Three other suggestions of the Committee on Chemical Agents deserve reiteration and discussion. It was urged that the bases for the selection of each value should be published, that the name should be changed to *hygienic standard,* and that the particular concept of permissible human response behind each value should be clearly indicated.

There is such a multitude of factors involved in the protection of health in our complex civilization that no one person or group of persons is competent to weigh them all with assurance. No oracular or *ex cathedra* statement on health deserves serious attention. Only when the facts upon which a decision are based are furnished for general scrutiny and evaluation can the decision be considered even tentatively sound, and only after there has been adequate opportunity for criticism and modification can it be considered established. All toxicological facts should be published, and all decisions upon the facts should be accompanied by a summary of the reasoning under which they were derived, before anyone should be expected to act upon the decisions. Any publication of standards for maintenance of health ought to include reference to the underlying data.

The second suggestion referred to the name by which the values are known. Semantics is more than a sport for the idle. No matter how thoroughly a concept is originally presented, it always becomes known and referred to by a brief name, a catchword. Most persons who learn of the concept hear the catch-word name, and do not go back to the original presentation. The meaning they attach to the name comes from their previous experience with the particular words. It may be, but is usually not, exactly what the originator of the idea intended. The more carefully one chooses the name he assigns to a concept, the more likely are others to interpret the concept as he himself does.

The values now known as threshold limits are usually identified by phrases containing the words *allowable* or *permissible.* These two words have connotations of legal regulations. Such connotations cannot properly attach to the judgment of a voluntary professional association. The identifying phrases may also contain the words *maximum, threshold* and *limit.* These words all

imply that below the concentration specified, human response is negligible, above the concentration it is dangerous. Actually, it is more than an implication. It is definitely stated. In the introduction to its 1956 list (ACGIH 1956) the Committee on Threshold Limits says, "Values are given . . . for the maximum average atmospheric concentrations of contaminants to which workers may be exposed for an eight-hour working day without injury to health." Careful study of the data which support the currently accepted values suggests that no such description can be truthfully attached to most of them. Industrial hygienists recognize this. They are accustomed to emphasize that the values should be regarded simply as bench marks, guides to good practice. Indeed, the Threshold Limits Committee itself confusingly warns "Threshold limits . . . should not be regarded as fine lines between safe and dangerous concentrations." (ACGIH 1956)

The term *maximum acceptable concentration* being used in revisions of standards by the American Standards Association Z-37 Committee is objectionable only because it will be abbreviated M.A.C. Many will interpret this abbreviation as *maximum allowable concentration,* and nothing will have been gained by the change from *allowable* to *acceptable.*

I conclude that the names *maximum allowable concentration* and *threshold limit* are misleading. They convey a wrong impression to those who are not already familiar with the concepts behind the values. The name suggested in 1949, *hygienic standard,* is not misleading. Standards of good practice are familiar to all of us in many fields. Looking toward the future provision of a variety of hygienic standards, a series of values should be selected, to be known as *hygienic standards for daily inhalation.*

The third suggestion is more far-reaching. The Committee on Chemical Agents (1949) pointed out that there has been no simple or uniform relation between the effects of a substance and the numerical value chosen for tabulation. The Committee concluded that concentrations have been selected on the basis of one of four concepts of the level best suited to hygienic control of inhalation, the choice having been governed by the nature of the toxic response and by the degree of organoleptic response. The Committee's four concepts follow:

a. *Plus or minus*: The maximal time-weighted average concentration which produces only minor injury, and that in a very small proportion of exposed workmen.

b. *Safe*: The maximal time-weighted average concentration which sound evidence leads one to believe will cause no demonstrable illness or other symptom of toxic effect in any workman during a lifetime of industrial exposure.

c. *Bench mark*: A concentration based on the belief that any unnecessary exposure is undesirable—a concentration lower than that of *a* or *b*, one as low as is consistent with practical engineering control.

d. *Comfort*: A concentration lower than *a* or *b*, representing the maximum which in a short time is not objectionable to 9 out of 10 of a group of persons not accustomed to inhalation of the substance.

Note well that these four concepts were judged to be those already used for the selection of hygienic standards for daily inhalation. All four were judged consistent with the goals of industrial hygiene.

Hygienic Standards for Daily Inhalation

THE SUBJECT of hygienic standards for daily inhalation should be re-examined, the concepts represented by the values should be restated in more realistic toxicological terms, and more consistent and more informative standards should be prepared. Such a step will not undo any of the accomplishments of the profession of industrial hygiene or of any organization. Rather, it will supply informative standards to supplement the accumulation of naked numbers now accepted, some of which have not been critically re-examined for a decade.

It is certainly imperative that the inhalation of substances during the working day shall not be allowed to result in any injury to the physical well-being of workmen. It is furthermore imperative that inhalation shall not increase the probability of accidents through the mental distress occasioned by objectionable eye, nose or throat irritation, transient though it may often be, nor through the impaired judgment and delayed reaction time of light narcosis. It is desirable that inhalation shall result in no degree of discomfort whatsoever. On the

other hand, when it is impractical to avoid all discomfort, then such inhalation is certainly justified, provided there results no injury to workmen and no increase in the probability of accidents.

Tables of hygienic standards do not now carry indications of the nature and of the magnitude of the effects to be expected from inhalation of greater concentrations. It is only by a rather thorough study of the available data that one can decide whether or not a particular substance can safely be inhaled at a greater concentration. With most substances, it is quite practical to set two standards, one an inoffensive level, another a concentration which cannot safely be exceeded under any pressure of practicality.

Administrative expediency may be served by a table of numbers which constitute a part of official regulations. Regulations need not be defended, they need not cite justifications. However, it is a minority of the profession of industrial hygiene who have regulatory responsibilities. Most of our colleagues act through obtaining voluntary cooperation with their judgments. They would be aided by a greater degree of explanation in a tabulation of standards. They could then show that their recommendations are quite defensible, that they are not arbitrary decisions having no regard for the realities of competitive industrial existence.

A hygienic standard for daily inhalation should specify two concentrations, together with a description of the human response to be expected from inhalation of each. One concentration should be low enough so that no injurious effect can be expected in any workman, but it may have a detectable odor, it may cause a detectable eye, nose or throat irritation. The second concentration should produce somewhat more severe, but still reversible and non-progressive effects. From these two concentrations, one can at once see how strictly the standard should be observed, the steepness of the dosage-response curve, the breadth of the plateau of non-injurious concentrations. Despite the wide range in individual susceptibilities of workmen, such values can be selected for most, if not all, substances.

Adequate data for preparing this sort of standard consist of appropriate human experience, or repeated medical examinations of workmen in atmospheres whose concentration has been frequently estimated. Sterner (1955) discussed this point in detail. Experiments upon animals, verified by biochemical and physiological studies in humans, may demonstrate conclusively that a substance has an effect during one period of inhalation which does not progress during oft-repeated inhalation. For such a substance, satisfactory data can be obtained from objective study and secretly recorded subjective effects of a group of humans inhaling known concentrations for substantially eight hours. Any lesser body of data should result in a hygienic standard being designated as tentative.

Categories of Objectionable Action

JUDGMENTS should be made to determine which hygienic standards for daily inhalation must be carefully observed, and which may be exceeded when it is impractical to observe them. These judgments will be most consistent if we first decide for each substance what objectionable action we are guarding against by the standard. Every toxicologist will realize that the action at a low concentration which it is most important to guard against, may not be the same as the menace to life to be expected at a high concentration. In a tentative fashion, the writer has made these decisions for the 238 substances, exclusive of the mineral dusts, included in the 1956 tables of proposed accepted and tentative standards (ACGIH 1956). The decisions can be divided into the following nine categories on the basis of the nature of human response.

Chronic toxicity. The most dangerous effect of some substances is a progressive systemic injury, increasing in severity with continuing inhalation. Benzene, carbon disulfide, carbon tetrachloride and lead are the most familiar examples. The lower standard for these substances should be a concentration believed not to produce any effect in any workman, and no considerations of practicality are sufficient to justify inhalation in excess of the standard. Close medical supervision is required for safe use of these substances. The standard for chronically toxic substances should refer to the time-weighted average concentration throughout a working day. Brief peaks of a few times the standard have no significance, save as they increase the average.

Acute toxicity. Some substances do not produce an injury progressing with repeated inhalation. Such systemic injury as they may cause takes place as the result of one excessive inhalation, or not at all. Familiar examples are carbon monoxide and hydrogen cyanide. The standards for acutely toxic substances should be interpreted in the same light as those for chronically toxic substances.

Narcosis. The most dangerous effect of some substances is narcosis, which becomes anesthesia in its extreme stage. At a rather low concentration they induce accidents by impairing judgment and delaying reaction time. Familiar examples are ethyl alcohol, ethyl ether and gasoline. The lower standard for a narcotic substance should be a concentration which produces no detectable effect upon judgment and reaction time after eight hours inhalation. It should refer to the average concentration existing during some appreciable period of time, the length of which can be estimated from absorption and elimination data. No considerations of practicality can justify exceeding the standard for a narcotic substance.

Irritation. The most dangerous effect of some substances is irritation. Eye, nose and throat are irritated at a low concentration, the bronchi at a higher concentration, and fatal lung edema may be the result of inhaling an extreme concentration. The aldehydes, halogens and acids are familiar examples. Highly odorous substances may also be considered in this category. The lower standard for an irritant substance should be a concentration which is detectable, but is not objectionably irritating to the majority of unhardened subjects who are exposed for a substantial part of a working day. The higher standard should be set at a concentration which is well under one injuring bronchi or lungs, and which is justifiable when it is impractical to keep concentrations at the lower standard. Standards for irritating substances should refer to concentrations existing for even a brief period during a work day.

Asphyxiation. Some substances are inert in the body and can injure only by asphyxia at extremely high concentrations, excluding the oxygen of the atmosphere. Familiar examples are the fluorochloro refrigerants. The lower standard for these asphyxiants should be a nominal bench mark of good engineering practice, such as the 1000 ppm concentration now quoted. The inert nuisance dusts like iron oxide might well be placed in this same category, and the currently used bench mark of 15 mg./cu.m. seems an appropriate level. The standard should refer to the concentration existing during any brief period, but it should be recognized that higher concentrations are justified when it is impractical to keep below the standard.

Fume fever. The most important effect of some substances is a transient influenza-like condition known as fume fever. A familiar example is zinc oxide fume. The lower standard for a fume fever producer should be a concentration which will not produce that distressing but not menacing condition in any workman, and it should apply to an appreciable period, such as half an hour. No considerations of practicality can justify exceeding the standard for fume fever producing substances.

Eye pigmentation. The most important effect of two substances, quinone and hydroquinone, appears to be a slowly developing pigmentation of the sclera, which may reduce visual acuity, or even lead to blindness. The lower standard for these substances should be a concentration which produces no pigmentation after years of exposure, and it should refer to the time-weighted average concentration throughout the day. For a few days at a time, conditions of practicality should justify exceeding the standard.

Cancer. One substance is reasonably well established as a cause of respiratory tract cancer. This is nickel carbonyl. It appears probable that the minimum cancerigenic exposure will never be defined. At this time it is prudent to set the standard for a cancerigenic substance substantially at zero, as has already been done for nickel carbonyl, and no considerations can justify allowing the inhalation of any concentration which is avoidable.

Allergy. Some substances are known to sensitize an appreciable proportion of exposed workmen. They may produce distressing and menacing asthma-like attacks when a sensitized person inhales a low concentration. Examples are ethylene diamine and the diisocyanates. At this time there is no

rational experimental basis for defining a concentration which will not sensitize a susceptible workman, or one to which no previously sensitized workman will respond. Control of exposure to allergenic substances must rely heavily upon industrial medicine. After experience has allowed withdrawal of workmen susceptible to sensitization, the remaining resistant individuals can be protected by a hygienic standard for daily inhalation based upon irritation or systemic injury. Until it has been demonstrated that a particular group includes no susceptible workmen, no considerations can justify allowing inhalation of any concentration which is avoidable.

Interpretations of Accepted Values

NO NEW VALUES for standards are suggested at this time. The available data upon which the 238 values in the 1956 proposed list (ACGIH 1956) appear to be based have been studied. Table I is offered as an interpretation of these values, increasing the information they convey. It presents the familiar numbers, which give the engineer and the chemist an illusion of complete understanding. It also presents, in the form of abbreviations of self-evident meaning, some description of actions which gives the biologically and medically trained a feeling of confidence. The table is obviously too complex for great popularity. Nevertheless, every class of information listed is required by those who must apply the values. The data relied upon for the interpretations and some comments on their adequacy are summarized after the table.

All substances in the proposed 1956 threshold limits table (ACGIH 1956), except mineral dusts, appear in one alphabetical order. When a value is listed in units of milligrams per cubic meter, the letter *m* precedes the number. When a tentative value was proposed the letter *T* follows the number.

Following the threshold limit values is a column showing a personal judgment of the most serious effect of inhalation of a concentration somewhat higher than the threshold limit. These judgments allow one to decide whether the value should refer to the time-weighted average concentration or to peak concentrations, existing at any time during the day. In future tables of standards the decision should be clearly indicated in the table.

Three columns record personal judgments and estimates as to what responses may occur in some workman inhaling continuously, all day, the threshold limit, twice, and ten times the limit. It will be obvious from these entries that the values may not always define concentrations in which workmen will find no objectionable sensory effect, or even concentrations where no toxic symptoms will develop in any individual.

Next comes a column listing important injuries other than from inhalation of the substance itself, such as dangerous absorption through the skin, chemical burns of eye and skin, frequent allergic dermatitis, pyrolysis to phosgene, and the like. The almost universal defatting of the skin by solvents, and freezing of tissue by low boiling liquids, has not been entered.

The last two columns give some indication of the soundness of the value by describing the supporting data, and by specifying the year in which it was first proposed or adopted. It is, of course, true that a value proposed many years ago and recopied in each succeeding year's list is not necessarily proven sound, but in general it is likely to be better established than a more recently adopted value.

There may be objection that the table does not mention warning power nor attempt to evaluate this property specifically. The practical importance of warning power in preventing inhalation of an excess is much over-rated. Odor data are notoriously unreliable. Estimates of tolerable working conditions with unacclimated subjects, briefly exposed, have only limited usefulness in predicting the responses of acclimated and usually hardened workmen, exposed all day. Early stages of narcosis reduce perception of odor and irritation. Even with strong irritants like ammonia and acrolein, physical circumstances, or a sense of duty, may keep a man at his post to be seriously injured by a concentration which, all would predict, cannot be inhaled voluntarily.

There is nothing in Table I which is not easily accessible, if not already well known to a thousand experienced industrial hygienists and toxicologists. Not one of these but will object to some among the thousand personal judgments entered. However, the

annual threshold limit tables are consulted by upwards of twenty thousand other persons who do not have access to original sources and extensive experience. These people rightly regard the threshold limits as a presentation of the best available judgment, and they may wrongly regard them as everything they need to know about safe use of a substance. Despite specific disclaimers, printed with each year's table, these readers tend to regard each threshold limit as defining the line between safety and injury.

Table I is presented as a beginning in the extensive job of improving communication by developing a rational and informative series of hygienic standards for daily inhalation.

Summary of Underlying Data

THE DATA relied upon for the interpretations entered in Table I are summarized and briefly commented upon in the pages which follow. It is believed that the most significant published information is included, but no attempt has been made to list all pertinent articles. Certain hitherto unpublished observations are included when they appear to confirm or supplement material in the literature. The term "most important effect" used with every substance, refers to the possible effect of inhalation of concentrations a few times the threshold limit, not necessarily to the possible effects at very high concentrations.

Acetaldehyde. Cook (1945) quotes the 1911 report of Iwanoff that cats inhaling 280 ppm for seven hours were not noticeably affected. The unacclimated subjects of Silverman, Schulte and First (1946) found 25 ppm objectionable, 50 ppm irritating to the eyes, but even 200 ppm not irritating to the throat. Fairhall (1949, p. 199) describes the effects as irritation, narcosis, bronchitis, albuminuria, fatty liver and lung edema. He concludes that inhalation does not cause chronic poisoning, and that death is due to anesthesia when prompt, or to lung edema when delayed. Smyth (1937-55) found rats survive four hours inhalation of 8000 ppm but die from 16,000 ppm. The liquid causes severe corneal injury, irritates the skin and may sensitize some persons.

MNEMONIC ABBREVIATIONS USED IN TABLE I.

acne-	*chloracne from continued skin contact.*
acute—	acute toxicity, with little or no increase in severity from continued inhalation.
all	allergenic. Dermatitis and asthma-like sensitization may result.
ALL—	allergenic. Dermatitis and asthma-like sensitization are likely.
asphyxia—	asphyxiation at very high concentrations.
bur—	burn of the skin.
BUR—	very severe burn of skin.
cancer—	cancer reported in humans.
chronic—	chronic toxicity, with increase in severity from continued inhalation.
clin—	clinical examination of workmen was correlated with their exposure.
cns—	central nervous system stimulation, such as tremors or convulsions.
cy—	cyanosis (blue skin) may be evident.
CY—	cyanosis (blue skin) may be marked.
est—	estimate from experience and analogy.
eye—	eye irritation severe enough to require medical treatment.
EYE—	eye burn may be severe.
eye pig—	eye pigmentation without injury.
fume—	fume fever.
head—	headache. This symptom has not been entered every time it may occur.
hu—	human sensory data.
HU—	human toxicological or physiological data.
ind—	industrial complaints or observations, less quantitative than clinical examinations.
irr—	irritation of eye, nose or throat in some.
IRR—	irritation of eye, nose or throat marked.
lung?-	minor irritation of bronchi (coughing) or lungs.
lung—	definite irritation of bronchi or lungs with injury of lungs possible.
LUNG—	dangerous injury of lungs with little warning.
m—	the quantity is expressed in milligrams per cubic meter (mg./cu.m.), not in ppm.
med—	medical uses yield some information.
narcosis—	narcosis, ranging from impaired coordination through dizziness, to anesthesia.
nar?—	faint narcosis, somewhat impaired reaction time and judgment.
nar—	narcosis definite, short of dizziness.
NAR—	narcosis marked, dizziness to unconsciousness.
nau—	nausea. This symptom has not been entered every time it may occur.
none—	no effects are expected.
odor—	odor may be perceptible.
ODOR—	odor marked.
pyr—	pyrolysis to lung injuring halogen compounds in a flame, or on hot metal.
rad—	radiation injury is possible.
rpt—	repeated animal inhalation results.
sgl—	single animal inhalation results.
skp—	skin penetration may cause symptoms.
SKP—	skin penetration of liquid is dangerous.
T—	tentatively proposed.
tox?—	toxic symptoms may arise very slowly.
tox—	minor toxic symptoms.
TOX—	major toxic symptoms.
vis—	visual acuity loss

TABLE I.
INTERPRETATION OF THRESHOLD LIMIT VALUES PROPOSED FOR 1956
Exclusive of Mineral Dusts
(Mnemonic abbreviations explained at foot of table)

Substance	Threshold Limit ppm or mg./cu.m.	Most Important Effect of Inhalation	Predicted Effects of Daily eight-hour Inhalations: At Threshold Limit	Additional At Twice Threshold Limit	Additional At Ten Times Threshold Limit	Important Hazards Other Than from Inhalation	Nature of Interpretive Data	Year Proposed
Acetaldehyde	200.	lung	IRR-odor		lung	all-EYE	hu-sgl	1945
Acetic acid	10.	lung	irr-odor		IRR-lung	bur-EYE	hu-ind-sgl	1945
Acetic anhydride	5.	lung	irr-odor		eye-IRR-lung	bur-EYE	sgl	1947
Acetone	1000.	narcosis	irr-nar-odor		NAR		HU-ind	1953
Acrolein	0.5	LUNG	irr	IRR	lung	bur-EYE	hu-sgl	1945
Acrylonitrile	20.	acute	none	odor	*tox*	*skp*	*rpt*	1943
Aldrin	m-0.25	chronic	none	none	irr-tox	all-skp	clin	1954
Allyl alcohol	5.	EYE-lung	irr		eye-lung	bur-EYE	ind-sgl	1954
Allyl chloride	5. T	lung	*none*		*irr-odor*	*pyr*	sgl	1956
Allyl propyl disulfide	2.	lung	irr	IRR			ind	1954
Ammate	m-15.	lung	none		lung		est	1954
Ammonia	100.	lung	irr-odor		IRR-lung	*bur-eye*	HU-ind-sgl	1943
Amyl acetate	200.	narcosis	irr-odor	IRR-nar	NAR		hu-sgl	1945
Amyl alcohol	100.	narcosis	irr-odor	IRR-nar	NAR		hu-sgl	1945
Aniline	5.	chronic	none	cy	CY-tox	SKP	rpt	1943
Antimony	m-0.5	chronic	none	none	tox		clin-rpt	1948
ANTU	m-0.3 T	chronic	none		tox		est	1956
Arsenic	m-0.5	chronic	none	tox?	tox		clin-est	1948
Arsine	0.05	acute-lung	none		lung ?-tox		ind-sgl	1947
Barium (soluble)	m-0.5	acute-lung	none		tox		est	1943
Benzene	35.	chronic	tox?	tox	nar-odor-**TOX**		*clin-rpt*	1948
Benzyl chloride	1.	lung	irr	odor	IRR-lung	EYE	sgl	1954
Bromine	1.	*lung*	*irr-odor*		*IRR-lung*	*BUR-EYE*	*ind-sgl*	1945
Butadiene	1000.	narcosis	none	odor	nar		hu-rpt	1947
Butanone (*methyl* ethyl ketone)	250.	narcosis	irr-odor	IRR-nau	nar		hu-sgl	1948
Butyl acetate	200.	narcosis	irr-odor	IRR-nar	NAR		hu-sgl	1945
Butyl alcohol	100.	narcosis	odor	irr	IRR-NAR		clin	1950
Butyl amine	5.	lung	odor	irr	eye	BUR-EYE	ind-sgl	1955
Butyl CELLO-SOLVE	200.	chronic	odor-tox?	nar-tox	irr-nar-**TOX**	skp	rpt	1945
Butyl mercaptan	10. T	lung	ODOR		eye-irr-lung?		sgl	1954
Cadmium oxide fume	m-0.1	acute-lung					ind-rpt	1943
Calcium arsenate	m-0.1 T	chronic	none	none	none		est	1956
Carbon dioxide	5000.	asphyxia	none		nar		HU	1943
Carbon disulfide	20.	chronic	none	odor	nar-tox		clin-rpt	1943
Carbon monoxide	100.	acute	none	tox	TOX		clin-HU	1943
Carbon tetrachloride	25.	chronic	tox?	tox	nar-odor-TOX	pyr	clin-ind-rpt	1953
CELLO-SOLVE	200.	chronic	odor	nar-tox	irr-nar-tox		rpt	1945
CELLO-SOLVE acetate	100.	chronic	odor	irr-tox	IRR-nar-tox		rpt	1945

TABLE I—CONTINUED

Substance	Threshold Limit ppm or mg./cu.m.		Most Important Effect of Inhalation	Predicted Effects of Daily eight-hour Inhalations: At Threshold Limit	Additional At Twice Threshold Limit	Additional At Ten Times Threshold Limit	Important Hazards Other Than from Inhalation	Nature of Interpretive Data	Year Proposed
Chlordane	m-2.0		chronic	none	none	irr-tox	all-skp	clin	1954
Chlorinated camphene (60%)	m-0.5	T	chronic	none		tox	all-skp	est	1956
Chlorinated diphenyl oxide	m-0.5		chronic	none	tox?	tox	acne	rpt	1955
Chlorine	1.		lung	odor	irr	IRR-lung	bur-EYE	hu-rpt	1948
Chlorine trifluoride	0.1		lung	none		irr-lung	bur-EYE	rpt	1955
Chlorobenzene	75.		narcosis	odor	irr-tox	nar	pyr	est-sgl	1948
Chlorobromomethane	400.	T	narcosis	none	nar	tox	pyr	rpt	1956
Chlorodiphenyl (42% Cl)	m-1.		chronic	none	none	tox?	acne	rpt	1945
Chlorodiphenyl (54% Cl)	m-0.5	T	chronic	none	tox?	tox	acne	rpt	1956
Chloroform	100.		chronic	odor-tox?	nar-tox	TOX	pyr	est-med-sgl	1945
1-Chloro-1-nitropropane	20.		lung	none		irr-lung-tox		sgl	1945
Chloropicrin	1.	T	lung	odor	irr	lung		clin	1956
Chloroprene	25.		chronic	none	nar-tox	TOX	pyr-skp	rpt	1945
Chromic acid, chromates as CrO_3	m-0.1		irr	none	irr	IRR	bur-EYE	clin	1943
CRAG herbicide	m-15.		chronic	none	none	irr-tox?		est	1954
Cresol	5.		chronic	odor	irr	lung-tox	BUR-EYE-SKP	est-sgl	1952
Cyanide as CN	m-5.		acute	none		tox		est	1947
Cyclohexane	400.		narcosis	none	nar-odor	irr-NAR-tox		rpt	1945
Cyclohexanol	100.		narcosis	irr-odor	nar-tox	IRR-NAR		hu-rpt	1945
Cyclohexanone	100.		narcosis	irr-odor		IRR-nar		hu-rpt	1945
Cyclohexene	400.		narcosis	none	nar-odor	irr-NAR-tox		sgl	1945
Cyclopropane	400.		narcosis	none	nar-odor	irr-NAR		est-med	1947
2,4-D	m-10.		chronic	none	none	irr-tox?		est	1954
DDT	m-1.	T	chronic	none	none	irr-tox	skp	est	1954
Decaborane	0.05	T	acute	none		odor		rpt	1956
Diacetone alcohol	50.		narcosis	irr	odor	IRR-nar		hu-sgl	1954
Diborane	0.1		cns-lung	none		lung-tox?		clin-rpt	1955
o-Dichlorobenzene	50.		chronic	odor-tox?	irr-tox	nar	pyr	ind-sgl	1947
Dichlorodifluoromethane	1000.		asphyxia	none	none	none	pyr	rpt	1947
1,1-Dichloroethane	100.		chronic	odor		nar-tox	pyr	rpt	1945
1,2-Dichloroethylene	200.		narcosis	none	odor	irr-nar	pyr	sgl	1945
Dichloroethyl ether	15.		lung	none	irr-odor	lung	skp	hu-sgl	1945
Dichloromonofluoromethane	1000.		asphyxia	none	none	none	pyr	sgl	1947
1,1-Dichloro-1-nitroethane	10.		lung	none	irr	lung-tox		rpt	1945

TABLE I—CONTINUED

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Dichlorotetrafluoromethane	1000.	chronic	none	none	none	pyr	sgl	1947
Dieldrin	m-0.25	asphyxia	none	none	irr	all-skp	clin	1954
Diethylamine	25.	lung	odor	eye-irr	IRR-lung	bur-EYE	sgl	1952
Difluorodibromomethane	100.	chronic	none	none	irr-nar-tox	pyr	rpt	1955
Diisobutyl ketone	50.	narcosis	irr-odor		IRR-nar		hu-rpt	1954
Diisocyano toluene	0.1 T	ALL-lung	all		ALL		ind	1956
Dimethylaniline	5.	chronic	none	cy	CY-tox	SKP	sgl	1943
Dimethylsulfate	1.	acute-LUNG	none		LUNG-TOX	BUR-EYE-SKP	sgl	1945
Dinitrobenzene	m-1. T	chronic	none		tox	skp	est	1956
Dinitro-o-cresol	m-0.2	acute	none		irr-tox	skp	sgl	1949
Dinitrotoluene	m-1.5	chronic	tox?	tox?	irr-tox	skp	est	1943
Dioxane	100.	chronic	none	nar-odor	irr-NAR-tox		hu-rpt	1947
EPN	m-0.5	acute	none		tox		est	1954
Ethyl acetate	400.	narcosis	irr-odor	nar	IRR-NAR		hu-sgl	1945
Ethyl acrylate	25. T	lung	odor	irr	lung		rpt	1956
Ethyl alcohol	1000.	narcosis	irr-odor	nar	IRR-NAR		HU	1945
Ethylamine	25.	lung	odor	eye-irr	IRR-lung	bur-EYE	sgl	1952
Ethyl benzene	200.	narcosis	nar?-odor	irr	NAR		hu-sgl	1945
Ethyl bromide	200.	lung	none	irr-odor	lung	pyr	sgl	1947
Ethyl chloride	1000.	narcosis	odor		nar	pyr	sgl	1947
Ethylene chlorhydrin	5.	acute	none	tox?	odor-tox	SKP	ind-sgl	1947
Ethylene diamine	10.	ALL-lung	odor	irr	eye	ALL-bur-EYE	ind-rpt	1955
Ethylene dibromide	25.	chronic-lung	none	irr-odor	lung-tox	pyr	rpt	1953
Ethylene dichloride	100.	chronic	odor-tox?	nar-nau	TOX	pyr	ind-rpt	1953
Ethylene imine	5.	acute-lung	odor		lung-TOX	bur-eye-SKP	hu-sgl	1955
Ethylene oxide	100.	lung-narcosis	odor-nar	irr	IRR-LUNG-NAR	bur	hu-rpt	1945
Ethyl ether	400.	narcosis	irr-odor	IRR	NAR		HU-med	1947
Ethyl formate	100.	narcosis	none	irr-odor	nar		sgl	1947
Ethyl mercaptan	250. T	lung	ODOR		eye-irr-lung		sgl	1954
Ethyl silicate	100.	acute-lung	odor	irr-tox	IRR		hu-rpt	1945
Ferbam	m-15. T	lung	none	irr	IRR		est	1956
Ferro vanadium dust	m-1.	lung	none		none	all	rpt	1954
Fluoride dust	m-2.5	chronic	tox?		irr-tox		clin-HU	1947
Fluorine	0.1	lung	none		irr-lung?-tox?	BUR-EYE	clin-rpt	1953
Fluoroacetates	m-0.1 T	acute	none		tox		est	1956
Fluorotrichloromethane	1000.	asphyxia	none	none	none	pyr	sgl	1945
Formaldehyde	5.	lung	irr	odor	IRR-lung	all-EYE	ind-sgl	1948
Furfural	5. T	lung	irr		IRR-lung	all-EYE	ind-sgl	1954
Furfuryl alcohol	50. T	narcosis	odor	irr-nar	IRR-NAR		rpt	1956
Gasoline	500.	narcosis	odor	irr-nar	NAR		hu-ind	1945

TABLE I—CONTINUED

Substance	Threshold Limit ppm or mg./cu.m.	Most Important Effect of Inhalation	Predicted Effects of Daily eight-hour Inhalations: At Threshold Limit	Additional At Twice Threshold Limit	Additional At Ten Times Threshold Limit	Important Hazards Other Than from Inhalation	Nature of Interpretive Data	Year Proposed
Heptane	500.	narcosis	odor	irr-nar	NAR		hu	1945
HETP	m-0.1	T acute	none		tox	SKP	est	1956
Hexane	500.	narcosis	odor	irr	irr-nar-nau		hu	1947
Hexanone (methyl butyl ketone)	100.	narcosis	odor	irr	nar		hu-sgl	1947
Hexone (methyl isobutyl ketone)	100.	narcosis	odor	irr	nar		hu-sgl	1947
Hydrazine	1.	lung	none		cns-irr-lung	bur-eye	rpt	1955
Hydrogen bromide	5.	lung	irr	IRR	eye-lung	bur-eye	hu	1955
Hydrogen chloride	5.	lung	irr	IRR	eye-lung	bur-eye	ind-rpt	1948
Hydrogen cyanide	10.	acute	odor		TOX	SKP	hu-sgl	1948
Hydrogen fluoride	3.	chronic-lung	irr	IRR	eye-lung-tox	BUR-EYE	ind-rpt	1943
Hydrogen peroxide, 90%	1.	lung	none		irr-lung	BUR-EYE	rpt	1955
Hydrogen selenide	0.05	chronic-lung	none		lung-odor-tox		hu-ind-rpt	1948
Hydrogen sulfide	20.	lung	irr-odor		lung-tox		clin-sgl	1943
Hydroquinone	m-2.	eye pig	none	eye pig	vis		clin	1955
Iodine	0.1	lung	irr	IRR	eye-lung	bur-eye	ind	1948
Iron oxide fume	m-15.	fume-lung	none	fume-irr	lung		clin	1947
Isophorone	25.	chronic-narcosis	irr-odor		IRR-nar-tox		hu-rpt	1945
Isopropylamine	5.	lung	odor	irr	eye	BUR-EYE	ind-sgl	1955
Lead	m-0.15	chronic	tox?	tox	TOX		clin	1943
Lead arsenate	m-0.15	T chronic	none	none	irr		rpt	1956
Lindane	m-0.5	chronic	none	none	irr	all-skp	rpt	1954
Magnesium oxide fume	m-15.	fume	none	fume-irr	lung		HU	1945
Malathon	m-15.	acute	none		tox		est-clin	1954
Manganese	m-6.	chronic	none	none	tox		clin	1945
Mercury	m-0.1	chronic	none	tox	TOX		clin-rpt	1943
Mercury, organic	m-0.01	chronic	tox?		tox	SKP	clin-sgl	1954
Mesityl oxide	50.	narcosis	irr-odor	nar	IRR-lung		hu-rpt	1945
Methoxychlor	m-15.	chronic	none	irr-tox			sgl	1954
Methyl acetate	200.	narcosis	odor	irr	nar		est-sgl	1948
Methyl acetylene	1000.	lung	none	odor	lung-nar		rpt	1955
Methylal	1000.	chronic	odor	irr	lung-tox		rpt	1952
Methyl acrylate	10.	T lung	none	odor	irr-nar		rpt	1956
Methyl alcohol	200.	narcosis	none	odor	irr-nar-tox?		rpt	1943
Methyl bromide	20.	chronic	none	cns-odor	irr-tox	pyr	clin-rpt	1945
Methyl CELLOSOLVE	25.	chronic	none	odor-tox?	irr-nar-tox		clin-rpt	1947

TABLE I—CONTINUED

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Methyl CELLOSOLVE acetate	25.		chronic	none	odor-tox	irr-nar-tox		est	1947
Methyl chloride	100.		chronic	none	odor	cns-nar	pyr	rpt	1947
Methyl chloroform	500.		narcosis	odor	nar	NAR	pyr	rpt	1953
Methyl cyclohexane	500.		narcosis	odor	nar	irr-NAR-tox		rpt	1947
Methyl cyclohexanol	100.		narcosis	irr-odor	nar-tox	IRR-NAR		rpt	1945
Methyl cyclohexanone	100.		narcosis	irr-odor		IRR-nar		rpt	1945
Methylene chloride	500.		chronic	none	odor	nar	pyr	rpt	1945
Methyl formate	100.		narcosis	none	irr-odor	nar		sgl	1947
Methyl isobutyl carbinol (methylamyl alcohol)	25.		narcosis	none	irr-odor	IRR-nar		hu-sgl	1954
Methyl mercaptan	50.	T	lung	ODOR		eye-irr-lung?		sgl	1954
Molybdenum (soluble)	m-5.		chronic	none	irr-tox			rpt	1955
Molybdenum (insoluble)	m-15.		chronic	none	none	irr-tox		rpt	1955
Naphtha (coal tar)	200.		narcosis	irr-nar?-odor		NAR		est	1945
Naphtha (petroleum)	500.		narcosis	odor	irr-nar	NAR		est	1945
Nickel carbonyl	0.001		cancer-lung	none	none	none		ind-sgl	1954
Nicotine	m-0.5	T	chronic	none		tox	SKP	est	1956
Nitric acid	10.	T	lung	irr	IRR	lung	BUR-EYE	est	1956
p-Nitroaniline	1.		chronic	none		tox	skp	est-ind	1954
Nitrobenzene	1.		chronic	none		tox	SKP	est	1947
Nitroethane	100.		acute	none	irr-odor	nar-tox		rpt	1947
Nitrogen dioxide	5.		lung	odor	irr	LUNG		ind-rpt	1945
Nitroglycerine	0.5		acute	head		tox	SKP	clin	1945
Nitromethane	100.		acute	none	irr-odor	nar-tox		rpt	1947
2-Nitropropane	50.		acute	nau-tox?		odor-tox		clin-sgl	1947
Nitrotoluene	5.		chronic	none	tox?	tox	SKP	est	1943
Octane	500.		narcosis	odor	irr-nar	NAR		est	1945
Ozone	0.1		lung	odor		irr-lung		rpt	1954
Parathion	m-0.1		acute	none		tox	SKP	clin-est	1953
Pentaborane	0.01	T	acute	none		none		rpt	1956
Pentachloronaphthalene	m-0.5		chronic	none	tox?	tox	acne	rpt	1945
Pentachlorophenol	m-0.5		acute	none		irr-tox	bur-skp	est	1947
Pentane	1000.		narcosis	odor	irr	nar		hu	1947
Pentanone (methyl propyl ketone)	200.		narcosis	irr-odor	IRR	nar		hu-sgl	1947
Perchloroethylene	200.		narcosis	odor	nar	irr-NAR	pyr	med-rpt	1953
Perchloromethyl mercaptan	0.1	T	lung	none		irr	bur-EYE	sgl	1954

TABLE I—CONTINUED

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Phenol	5.	chronic	odor	irr	lung-tox	BUR-EYE-SKP	hu-rpt	1952
Phenylhydrazine	5.	*chronic*			*cy*-TOX	all-SKP	sgl	1954
Phosgene	1.	lung	none		LUNG-odor	bur-EYE	HU-rpt	1943
Phosphine	0.05	chronic	none		none		rpt	1947
Phosphorous (yellow)	m-0.1	chronic	none		tox?		ind	1947
Phosphorous pentachloride	m-1.	lung	none		irr-lung	bur-EYE	sgl	1947
Phosphorous pentasulfide	m-1.	lung	none		irr-lung		est	1947
Phosphorous trichloride	0.5	lung	none	irr	lung-odor	bur-EYE	sgl	1945
Picric acid	m-0.1	chronic	none	irr	tox	all-skp	clin	1954
Propyl acetate	200.	*narcosis*	*irr-odor*	IRR-*nar*	NAR		*est-sgl*	1945
Propyl alcohol (isopropanol)	400.	narcosis	irr-odor	nar	IRR-NAR		est-hu	1945
Propylene dichloride	75.	chronic	tox?	nar-odor	TOX	pyr	rpt	1947
Propylene imine	25.	acute-lung	odor		lung-TOX	bur-EYE-SKP	sgl	1955
Propyl ether	500.	narcosis	irr-odor	IRR	NAR		rpt	1945
Pyrethrum	m-2. T	lung	none		irr		rpt	1956
Pyridine	10.	chronic	cns?-odor		irr-tox		ind-med	1954
Quinone	0.1	eye pig	none	eye-pig	vis		clin	1955
Rotenone	m-5. T	lung	none		irr		est	1956
Selenium compounds, as Se	m-0.1	chronic	none		tox		clin-est	1947
Sodium hydroxide	m-2.	irr	irr		IRR	bur-EYE	ind	1954
Stibine	0.1	chronic-lung	none		lung?-tox?		sgl	1947
Stoddard solvent	500.	narcosis	odor	irr-nar	NAR		hu	1945
Strychnine	m-0.15 T	acute	none		tox		est	1956
Styrene monomer	200.	narcosis	odor	irr	IRR-nar		hu-rpt	1947
Sulfur dioxide	10.	lung	irr-odor	IRR	lung		clin-hu	1943
Sulfur hexafluoride	1000.	asphyxia	none	none	none	pyr	sgl	1954
Sulfuric acid	m-1.	lung	irr		IRR-lung	bur-EYE	HU-ind-sgl	1948
Sulfur monochloride	1.	lung	none	irr	IRR-lung	bur-EYE	ind-sgl	1945
Sulfur pentafluoride	0.025	*lung*	*none*		irr-lung	bur-EYE	sgl	1954
TEDP	m-0.2	acute	none		tox		est	1954
Tellurium	m-0.1	chronic	none	none	irr-tox?		clin	1947
TEPP	m-0.05	acute	none		tox		est	1954
p-Tertiary butyl toluene	10.	chronic	odor		irr-nar-tox		hu-rpt	1955
Tetrachloroethane	5.	chronic	tox?	tox	odor-TOX	pyr	ind-sgl	1947
Tetrahydrofuran	200. T	narcosis	none	odor	irr		rpt	1956
Tetranitromethane	1.	acute	irr		tox	skp	rpt	1955
Tetryl	m-1.5	chronic	none	irr	tox	all-skp	clin	1943
Thallium (soluble)	m-0.15 T	chronic					est	1956

TABLE I—CONTINUED

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Thiram	m-5.	T chronic	none				est-sgl	1956
Titanium dioxide	m-15.	lung	none		irr-lung		rpt	1954
Toluene	200.	narcosis	nar?-odor	irr	NAR		clin-hu-rpt	1943
o-Toluidine	5.	chronic	none	cy	CY-tox	SKP	sgl	1945
Trichloro-ethylene	200.	narcosis	nar-nau-odor		irr-NAR	pyr	ind-med-rpt	1948
Trichloro-naphthalene	m-5.	chronic	none		tox	acne	rpt	1945
Trifluoromon-obromo-methane	1000.	narcosis	none	none	irr-nar	pyr	rpt	1955
Trinitrotoluene	m-1.5	chronic	tox?	tox?	irr-tox	skp	clin-rpt	1943
Turpentine	100.	narcosis	irr-odor	IRR-nar	NAR-tox		hu-rpt	1945
Uranium (soluble)	m-0.05	chronic	none		tox	rad	rpt	1953
Uranium (insoluble)	m-0.25	chronic	none		tox	rad	rpt	1953
Vanadium (V_2O_5 dust)	m-0.5	lung	none		lung		rpt	1954
Vanadium (V_2O_5 fume)	m-0.1	lung	none	lung			rpt	1954
Vinyl chloride	500.	narcosis	none		nar	pyr	sgl	1947
Warfarin	m-0.5	T chronic	none		tox		est	1956
Xylene	200.	narcosis	irr-nar?-odor	nar	NAR		hu	1943
Zinc oxide fume	m-15.	fume	fume	fume-irr	lung		HU	1943
Zirconium	m-5.	T lung	none		lung		rpt	1955

The most important effect of acetaldehyde inhalation is irritation of upper respiratory tract, bronchi and even lung. The 200 ppm threshold limit can be interpreted from human sensory data. It is sufficiently low to prevent lung injury.

Acetic acid. Sterner (1943) concludes 10 ppm is reasonably non-irritating on the basis of industrial experience. Patty (1948-9, p. 886) finds 800 to 1200 ppm intolerable. Smyth (1937-55) found inhalation of 16,000 ppm by rats for four hours killed one of six. The liquid causes severe corneal injury. Vigliani and Zurlo (1955) report workers exposed seven to 12 years to concentrations of 60 ppm, with one hour daily at 100 to 260 ppm, had no injury except slight irritation of the respiratory tract, stomach and skin. They regard 20 to 30 ppm as without danger.

The only important effect of acetic acid inhalation is irritation, first evident in the eyes, then in the upper respiratory tract, bronchi and even lung. The 10 ppm threshold limit can be interpreted from uncontrolled human sensory data. It is low enough to prevent lung injury.

Acetic anhydride. Henderson and Haggard (1943, p. 130) mention eye, nose and throat irritation and suggest that bronchial and lung injury are likely. Fairhall (1949, p. 203) considers it a lacrimator and finds systemic effects unlikely. McLaughlin (1946) discusses serious corneal injury from the liquid in industry. Smyth (1937-55) found rats inhaling 1000 ppm for four hours survived, but 2000 ppm was fatal. The liquid causes skin burns.

The only important effect of acetic anhydride inhalation is irritation, first evident in the eyes, then in the upper respiratory tract, bronchi and even lung. The 5 ppm threshold limit can be interpreted from analogy with acetic acid. In view of rat mortality from the two vapors, a lower value would be more consistent, although it is undoubtedly low enough to prevent lung injury.

Acetone. Nelson, Ege, Ross, Woodman and Silverman (1943) found slight eye,

nose and throat irritation with unacclimated subjects at 300 ppm, but 500 ppm was not objectionable. Henderson and Haggard (1943, p. 196) conclude death is anesthetic, with no organic injury below a narcotic level. Fairhall (1949, p. 205) concludes it causes narcosis, bronchial irritation and headache, but no chronic systemic effect. Haggard, Greenburg and Turner (1944) found human narcosis like that from ethyl alcohol. The highest concentration not causing narcotic impairment of coordination and judgment is 2110 ppm, which results in a blood level ⅓ that giving first alcoholic intoxication symptoms. Smyth's (1937-55) rats survived four hours at 32,000 ppm, died from 64,000 ppm. Vigliani and Zurlo (1955) found chronic respiratory tract irritation and dizziness in workers inhaling 1000 ppm three hours a day.

The most important effect of acetone inhalation is narcosis. The 1000 ppm threshold limit can be interpreted from human sensory and physiological data. It is not low enough to prevent all narcotic symptoms.

Acrolein. Yant, Schrenk, Patty and Sayers (1930) found marked human eye, nose and throat irritation within five minutes at 1 ppm. Patty (1948-9, p. 936) concludes 0.25 ppm is moderately irritating. Henderson and Haggard (1943, p. 138) conclude the main attack is on the upper respiratory tract, but that a high concentration can cause lung edema. They report 10 ppm to be lethal in a short time. Systemic effects are not to be expected. Smyth (1937-55) found four hours inhalation of 8 ppm kills one of six rats and all die from 16 ppm. The liquid causes severe corneal injury and burns of the skin.

The only important effect of acrolein inhalation is irritation, first evident in the eyes, then in the upper respiratory tract, bronchi and even lung. The 0.5 ppm threshold limit can be interpreted from human sensory data. It is low enough to prevent lung edema.

Acrylonitrile. Dudley, Sweeney and Miller (1942) found repeated inhalation of 153 ppm injurious to animals, but believed the effect not chronic toxicity. Dudley and Neal (1942) concluded injury is due to formation of cyanide in the body. The 10 ppm hydrogen cyanide threshold limit is equivalent to 20 ppm acrylonitrile, if conversion is complete. Wilson (1944) in exposed workmen, found evidence of skin penetration and effects referable to liver injury. Smyth's (1937-55) rats survived four hours at 500 ppm but were killed at 1000 ppm.

The most important effect of acrylonitrile inhalation is acute poisoning, due to hydrolysis in the body to cyanide. The 20 ppm threshold limit can be interpreted from results of repeated animal inhalation studies and its relationship to the accepted 10 ppm threshold limit for hydrogen cyanide. It is low enough to prevent injury.

Aldrin. Princi and Spurbeck (1951) examined workers with one to three years exposure to 1 to 2.6 mg./cu.m. aldrin and related dusts, and found no clinical evidence of injury. McGee (1955), reviewing human cases and animal data, finds aldrin and lindane have similar actions. Acutely they increase central nervous system irritability, leading to convulsions. Chronically they injure the liver, with effects also on kidney, lung and nervous system, and they sensitize some skins. ACGIH (1954b) finds aldrin twice as toxic to animals acutely as lindane, and concludes half the threshold limit of the latter is tentatively appropriate.

The most important effect of aldrin inhalation is chronic poisoning centering in the liver. The 0.25 mg./cu.m. threshold limit can be interpreted from the results of examination of exposed workmen. It is low enough to prevent injury.

Allyl alcohol. McCord (1932) found some human irritation at 5 ppm. The review by von Oettingen (1943, p. 138) shows cats die during 30 seven-hour inhalations of 50 ppm, with pulmonary edema, gastroenteritis, hematuria and nephritis. Smyth (1937-55) found rats survive one hour at 500 ppm, but die from 1000 ppm. The vapors irritate eye and nose, but not sufficiently so to prevent exposure to a concentration which temporarily blinded one man through delayed corneal necrosis. Chronic toxicity is not to be expected, but skin penetration is dangerous, and skin contact causes burns when evaporation is prevented.

The most important effect of allyl alcohol inhalation is irritation, manifest as disabling corneal injury and pulmonary edema, with non-progressive organic effects somewhat less important and narcosis overshadowed. The 5 ppm threshold limit can be

interpreted from a report of human irritation and results of repeated animal inhalation. It is probably low enough to prevent injury, but may be slightly irritating to some.

Allyl chloride. Adams, Spencer and Irish (1940) found rats survive three hours inhalation of 290 ppm, while eight hours killed all. Injury was chiefly in the lung, with some kidney effects. Narcosis was weak but mucous membrane irritation was prominent.

The most important effect of allyl chloride inhalation is irritation of the upper respiratory tract, bronchi and lung. The 5 ppm tentative threshold limit can be interpreted from single inhalations by animals and by analogy with chloroprene. It appears low enough to prevent injury.

Allyl propyl disulfide. Feiner, Burke and Baliff (1946) surveyed an onion dehydrating plant and found pronounced irritation of eye, nose and throat at 3.4 ppm onion oil calculated as this disulfide, with some irritation at 2 ppm. Acclimitization was evident.

The most important effect of allyl propyl disulfide inhalation is irritation of eye, upper respiratory tract and lung. The 2 ppm threshold limit can be interpreted from the results of complaints from exposed workmen. It appears low enough to prevent injury.

Ammate. ACGIH (1954b) concludes the single dose LD_{50} for animals of 2000 mg./kg. justifies no more control on inhalation than is required for a non-toxic nuisance dust.

The most important effect of ammate dust inhalation is the low grade irritation of a substantially inert dust. The 15 mg./cu.m. threshold limit can be interpreted only by analogy. It appears low enough to prevent injury.

Ammonia. Lehmann (1886) suggested 100 ppm is tolerable, and subsequent industrial experience has been favorable. Henderson and Haggard (1943, p. 125) note incapacitating temporary blindness, and respiratory arrest from a high concentration. They state 53 ppm is the least amount smelled, but Smyth (1937-55) found 1 ppm detected and identified by 10 subjects. They give 408 ppm as irritating to the throat, 698 ppm irritating to the eye, 2500 to 6500 ppm dangerous to life in 30 minutes. Fairhall (1949, p. 20) notes eye and upper respiratory tract irritation, salivation, bronchial irritation and lung edema, but no chronic systemic effect. Elkins (1950, p. 84) found 55 ppm not objectionable in industry but 125 ppm irritating. Silverman, Whittenberger and Muller (1949) found 500 ppm stimulated human respiration, irritated eye and throat, caused lacrimation. Smyth (1937-55) found rats survive four hours at 2000 ppm, die at 4000 ppm. Solutions irritate the skin, erode mucous membrane and severely injure the cornea. Vigliani and Zurlo (1955) in workers inhaling 100 ppm, found irritation of the respiratory tract and conjunctiva. Even 20 ppm caused complaints until workers became hardened.

The most important effect of inhalation of ammonia gas is respiratory tract irritation, with lung edema or respiratory arrest the maximum injury. The 100 ppm threshold limit can be interpreted from human sensory and physiological data. It is low enough to prevent injury.

Amyl acetate. Patty, Yant and Schrenk (1936) found 2000 ppm does not injure guinea pigs in several hours, while a concentration killing in 60 minutes can not be obtained. Symptoms consisted of eye and nose irritation and narcosis. Nelson, Ege, Ross, Woodman and Silverman (1943) found slight throat irritation in unacclimated subjects at 100 ppm, mild eye and nose sensation and severe throat irritation at 200 ppm. Smyth (1937-55) with rats inhaling substantially saturated vapors found anesthesia in two hours and death in eight hours. Chronic toxicity is not to be expected.

The most important effect of amyl acetate inhalation is narcosis. The 200 ppm threshold limit can be interpreted from human sensory data and single inhalations by animals. It is low enough to prevent definite narcosis.

Amyl alcohol (isoamyl alcohol.) Nelson, Ege, Ross, Woodman and Silverman (1943) found slight throat irritation in unacclimated subjects at 100 ppm, and objectionable eye, nose and throat irritation at higher concentrations. Haggard, Miller and Greenberg (1945) found the toxicity 12 times that of ethyl alcohol for anesthetic death. No chronic systemic toxicity is to be expected. Smyth (1937-55) found rats not

killed by eight hours at 2000 ppm, close to saturation.

The most important effect of amyl alcohol inhalation is narcosis. The 100 ppm threshold limit can be interpreted from human sensory data and analogy with butyl alcohol. It is low enough to prevent significant narcosis, but not to prevent slight irritation.

Aniline. Henderson and Haggard (1943, p. 227) conclude 7 to 25 ppm gives slight symptoms in several hours, 100 to 160 ppm for one hour causes serious disturbance. Aniline is a chemical asphyxiant, causing methemoglobin cyanosis through its metabolite, p-aminophenol. This can lead to anemia, but death from a single exposure is due to central nervous effects leading to respiratory paralysis. Skin penetration is more an industrial hazard than inhalation. Smyth (1937-55) found 340 ppm, substantial saturation, did not kill rats in two hours but was fatal in four, with their hemoglobin 54% converted to methemoglobin. Oberst, Hackley and Comstock (1956) found repeated inhalation of 5 ppm caused methemoglobinemia in rats but not any symptoms in dogs. They explained the difference in response by the fact that rats breathe three times as much air per unit time as do dogs, hence absorbed more aniline.

The most important effect of aniline inhalation is acute poisoning, in which cyanosis is evident but not of major importance. The 5 ppm threshold limit can be interpreted from results of repeated animal inhalations. It appears low enough to prevent injury.

Antimony. Bradley and Fredrick (1941) administered various antimony compounds orally and intraperitoneally to rats. They concluded it is more toxic than lead, but is not stored. The most important effect was on the heart muscle, and they advised that exposed workmen should be followed electrocardiographically. Dernehl, Nau and Sweets (1945) exposed guinea pigs to antimony oxide of one micron diameter at 45 mg./cu.m., three hours a day for several months. One-sixth died of pneumonitis with severe liver injury and white blood cell changes, but hearts remained normal. Electrocardigrams on the animals and on a few industrially exposed workmen were normal. Brieger, Senisch, Stasney and Piatnek (1945) in an industrial operation where antimony trisulfide concentrations ranged from 0.58 to 5.5 mg./cu.m., found abnormalities in blood pressure, electrocardiographic changes and two deaths from chronic thrombosis.

The most important effects of inhalation of antimony dust are chronic poisoning marked by electrocardiographic changes, pneumonitis and liver injury. The 0.5 mg./cu.m. threshold limit can be interpreted from the results of repeated inhalations and examination of workmen. It appears low enough to prevent injury.

ANTU (alphanaphthylthiourea). McClosky and Smith (1945) found considerable species differences in acute oral toxicity. The LD_{50} for rats, the most susceptible species, was about 0.03 gm./kg. Death was due to pleural efusion. Repeated doses caused liver injury. Fitzhugh and Nelson (1947) found rats are not affected by 50 ppm in their diet over a two-year period. Tolerance developed.

The most important effect of ANTU inhalation is chronic poisoning centering in the liver. The 0.3 mg./cu.m. tentative threshhold limit can be interpreted from the results of repeated oral doses to rats. It corresponds to a maximum daily absorption of three milligrams, about 0.05 mg./kg. This appears low enough to prevent injury.

Arsenic. The earlier limit of 0.15 mg./cu.m. was based upon supposed quantitative similarity to lead. Watrous and McCaughey (1945) found workers exposed to 0.007 to 0.60 mg./cu.m. showed no symptoms. Chronic arsenic poisoning causes varied symptoms (I.L.O., 1930, I, p. 161): digestive disturbance, hyperemia, skin eruption, pigmentation, keratosis, epithelial cancer, polyneuritis, cirrhosis, confusion, delirium, irritation of eyes and respiratory tract. Arsenic is stored in the body, but not to the extent of lead.

The most important effect of inhalation of arsenic compounds is chronic poisoning. The 0.5 mg./cu.m. threshold limit can be interpreted from the results of examinations of exposed workmen. It appears low enough to prevent injury.

Arsine. Henderson and Haggard (1943, p. 241) describe acute arsine poisoning as due to hemolysis of red blood cells with resulting anemia and kidney damage, and lung

edema. They state 250 ppm for 30 minutes is fatal, and 3 to 10 ppm can cause symptoms in a few hours. Nau (1948) reported an industrial episode showing the earlier limit of 1 ppm was too high, and Elkins (1950, p. 67) reported briefly on a non-fatal case at a level of about 0.5 ppm.

The most important effect of arsine inhalation is acute poisoning, largely lung edema. The 0.05 ppm threshold limit can be interpreted from the results of industrial experience. It appears to be low enough to prevent injury.

Barium (soluble compounds). Fairhall (1949, p. 32) records the fatal dose of soluble barium compounds as 0.8 to 0.9 grams with gastro-intestinal disturbance the chief symptom. He notes bronchial irritation from barium carbonate dust, and depilatory action of barium sulfide.

The most important effect of inhalation of soluble barium compounds is bronchial irritation, with acute poisoning possible. The 0.5 mg./cu.m. threshold limit can be interpreted only by analogy with antimony. It appears low enough to prevent injury.

Benzene. Winslow (1927) first proposed a limit of 100 ppm, based on extensive examination of exposed workmen and animal inhalation. He recognized that chronic poisoning would develop in some at this concentration, but believed it would progress slowly enough to be detected by periodic medical examinations, and arrested by removal from exposure. Acute benzene poisoning is fatal anesthesia, and chronic poisoning is primarily injury to the bone marrow. Benzene is particularly insidious because its effects can progress to a fatal outcome after all exposure ceases. Even brief inhalation of a high non-anesthetic concentration can be fatal. Patty (1948-9, p. 757) states that 100 ppm has only a faint odor. Elkins (1950, p. 228) investigated a fatal case whose exposure he was convinced had been only to 40 to 80 ppm.

The most important effect of benzene inhalation is chronic poisoning centering in the bone marrow. The 35 ppm threshold limit can be interpreted from extensive examination of exposed workmen, and was quantitatively defined by one fatal case. It appears low enough to prevent the development of irreversible poisoning.

Benzyl chloride. This is a potent lacrimator, irritating to eye, nose and throat, and capable of causing lung edema. Flury and Zernik (1931, p. 538) conclude 170 ppm is dangerous to cats in eight hours and 16 ppm intolerable to man in one minute. It may be inferred that the liquid causes severe corneal injury.

The only important effect of benzyl chloride inhalation is irritation, first evident in the eyes, then in the upper respiratory tract, bronchi and even lung. The 1 ppm threshold limit can be interpreted from older human sensory data. It is undoubtedly low enough to prevent lung injury.

Bromine. Flury and Zernik (1931) quote Lehmann that 0.75 ppm in a workroom caused no symptoms in six hours. Henderson and Haggard (1943, p. 133) state bromine acts as a respiratory irritant leading to lung edema. They state 40 to 60 ppm is dangerous on short inhalation, and 4 ppm allowable for 30 to 60 minutes. Elkins (1950, p. 87) found 1 ppm excessively irritating. Severe burns of skin and cornea result from the liquid. Patty (1948-9, p. 554) concludes 0.3 ppm is not objectionably irritating.

The most important effect of inhalation of bromine vapor is respiratory tract irritation, with lung edema the maximum effect. The 1 ppm threshold limit can be interpreted from industrial experience. It appears low enough to prevent injury.

Butadiene. Von Oettingen (1940) quotes repeated animal exposures at 64,000 ppm which caused bronchial and lung irritation and some hyperplasia of bone marrow. Carpenter, Shaffer, Weil and Smyth (1944) found animals not affected by repeated inhalation of 2300 ppm, while 6700 ppm slightly retarded growth and there were minor liver effects. Two humans found psychomotor effects of early narcosis from 8000 ppm, equivalent to those from 200 ppm toluene.

The most important effect of butadiene vapor inhalation is narcosis. The 1000 ppm threshold limit can be interpreted from results of repeated animal inhalation and single human inhalation. It is low enough to prevent any degree of narcosis.

Butanone (methyl ethyl ketone). Patty, Schrenk and Yant (1935) found guinea pigs tolerated 3000 ppm for several hours, and men found it irritating to nose and eyes. Nelson, Ege, Ross, Woodman and Silverman

(1943) found slight throat irritation in unacclimated subjects at 100 ppm, irritation of eyes at 200 ppm and objectionable irritation at 300 ppm. Elkins (1950, p. 118) found complaints of nausea at 500 ppm, irritation at 300 ppm, but no ill effects at 700 ppm. Smyth (1937-55) found rats survived two hours at 2000 ppm but 4000 ppm killed four of six. He found that an episode of industrial eye injuries during inhalation of butanone was caused by an unsaturated ketone impurity accidently present.

The most important effect of butanone inhalation is narcosis. The 250 ppm threshold limit can be interpreted from human sensory data. It appears low enough to prevent definite narcosis.

Butyl acetate. Sayers, Schrenk and Patty (1936) found guinea pigs are not affected by several hours inhalation of 3300 ppm. Nelson, Ege, Ross, Woodman and Silverman (1943) found throat irritation in unacclimated subjects at 200 ppm, severe at 300 ppm. Henderson and Haggard (1943, p. 222) conclude the ester shows no chronic toxicity. Smyth (1937-55) found rats inhaling substantially saturated vapors are not killed in four hours, but died within an eight-hour inhalation period.

The most important effect of butyl acetate inhalation is narcosis. The 200 ppm threshold limit can be interpreted from human sensory response and single inhalations by animals. It is low enough to prevent definite narcosis.

Butyl alcohol (n-butanol). Tabershaw, Fahy and Skinner (1944) reported eye inflammation in workmen above 50 ppm, but no systemic effects below 100 ppm. Sterner, Crouch, Brockmyre and Cusack (1949) followed workmen for 10 years with butyl alcohol concentrations held to 100 ppm, and for a briefer period to 200 ppm. Neither irritation nor systemic effects were found at 100 ppm, but there was some eye irritation at 200 ppm. Smyth (1937-55) found rats are not killed in four hours at 8000 ppm.

The most important effect of butyl alcohol inhalation is narcosis. The 100 ppm threshold limit can be interpreted from an extensive study of workmen under conditions of known peak exposure. No narcotic or irritative effects are to be anticipated.

Butyl amine. Hanzlik (1923) reported central nervous stimulation, convulsions, then depression and narcotic death with pulmonary edema. Smyth (1937-55) found rats survive four hours at 2000 ppm but die from 4000 ppm. Injury to skin and cornea from the liquid is severe. Unreported industrial experience suggests skin injury is the greatest practical hazard. ACGIH (1955b) cites unpublished industrial experience that levels above 5 ppm tend to be irritating.

The most important effect of butyl amine inhalation is respiratory tract irritation, with lung edema the maximum injury. The 5 ppm threshold limit can be interpreted from analogy with ethyl amine. It is probably low enough to prevent injury.

Butyl CELLOSOLVE (2-butoxyethanol). Werner, Nawrocki, Mitchell, Miller and von Oettingen (1943) found 300 to 400 ppm, in repeated inhalation produced only small effects on rats, particularly on the blood picture. Werner, Mitchell, Miller and von Oettingen (1943a, b) reporting on single inhalations by rats and repeated by dogs, make it clear that the butyl ether produces somewhat greater blood cell changes than do the methyl or ethyl ethers. They also found hemoglobinuria, with lung, liver and kidney changes. Smyth (1937-55) found in rats fractional mortality from as little as 500 ppm inhaled eight hours, with hematuria a prominent symptom. The liquid penetrates the skin readily and is sufficiently toxic so that this is dangerous.

The most important effect of butyl CELLOSOLVE inhalation is chronic poisoning, centering in the blood cells and kidney. The 200 ppm threshold limit can be interpreted from results of repeated animal inhalation studies. Based on reports from simultaneous studies of CELLOSOLVE and butyl CELLOSOLVE it is obvious that the threshold limit for the latter should be lower than for the former if equal degrees of protection are to be attained.

Butyl mercaptan. Fieldner, et al. (1931) reports 733 ppm to be lethal to dogs in 30 minutes, indicating 20 times the acute toxicity of ethyl mercaptan. Effects like those of hydrogen sulfide are to be expected.

The most important effect of butyl mercaptan is eye and respiratory tract irritation. The 10 ppm tentative threshold limit can be interpreted from the results of limited single inhalations by animals and analogy with hydrogen sulfide. It appears low

enough to prevent injury and eye irritation.

Cadmium oxide fume. Prodan (1932) on the basis of animal experiment concluded it is as toxic as lead. Spolyar, Keppler and Porter (1944) reported on serious poisoning and fatalities from industrial exposure to the fume. Fairhall (1949, p. 45) concludes inhalation causes bronchial irritation and pneumonitis, while ingestion produces gastro-intestinal disturbances.

The most important effect of inhalation of cadmium oxide fume is severe lung injury, with systemic poisoning less important. The 0.1 mg./cu.m. threshold limit cannot be interpreted quantitatively, but it appears low enough to prevent injury.

Calcium Arsenate. ACGIH (1954b) bases the tentative threshold limit upon a rat oral LD_{50} of 100 mg./kg., and blind litters in rats fed 5 mg./kg. for 45 days.

The most important effect of inhalation of calcium arsenate dust is chronic arsenic poisoning, with bronchial irritation less important. The 0.1 mg./cu.m. tentative threshold limit should be interpreted from the threshold limit for arsenic dusts. Since calcium arsenate is 20% arsenic, a threshold limit of 2.5 mg./cu.m. would be consistent with the accepted limit for arsenic dusts.

Carbon dioxide. Flury and Zernik (1931) quote Lehman-Hess to the effect that 5500 ppm causes no noticeable symptoms in six hours. Aero Medical Association (1953, p. 52) considers the gas as weakly narcotic, 30,000 ppm increasing respiration by 90%, increasing pulse and blood pressure, and decreasing acuity of hearing. Subjective symptoms arise above this level, 50,000 ppm for 30 minutes giving the first signs of intoxication, and 70,000 to 100,000 ppm causing unconsciousness in a few minutes.

The most important effect of carbon dioxide inhalation is asphyxia at very high concentrations. The 5000 ppm threshold limit can be interpreted from the results of extensive human experiments. It is low enough to prevent noticeable effects.

Carbon disulfide. Wiley, Hueper and von Oettingen (1936) found repeated inhalation of 30 ppm has no significant effect on animals. Barthelemy (1939) found no injuries to rayon workmen when concentrations were kept below 30 ppm. In single inhalations, it is markedly narcotic, and in repeated exposure the effects may be neurological, not recognized by the victim. Henderson and Haggard (1943, p. 223) state that 480 to 1600 ppm is the maximum breathable for one hour without serious disturbance.

The most important effect of carbon disulfide inhalation is chronic poisoning with central nervous system effects. The 20 ppm threshold limit can be interpreted from the results of repeated animal inhalations and examination of exposed workmen. It is low enough to prevent injury.

Carbon monoxide. Henderson, Haggard, Teague, Prince and Wunderlich (1931) suggested a limit of 100 ppm on the basis of extensive human experiment. Sayers, Yant, Levy and Fulton (1929) showed 200 ppm caused slight symptoms in humans. Sievers, Edwards, Murray and Schrenk (1942) found 70 ppm over a 13-year period had not affected health. Carbon monoxide is a chemical asphyxiant, acting by combining with hemoglobin. Henderson and Haggard (1943, p. 167) define its effects in terms of the product of time and concentration, 100 ppm for three hours producing no effect; for six hours, a just appreciable effect; for nine hours, headache and nausea; and for 15 hours, danger. One hour at 4000 ppm may be fatal. Vigliani and Zurlo (1955) studying 100 workers found no injury to health at 100 ppm for eight hours every day. In the United States chronic poisoning is not considered a reality.

The most important effect of carbon monoxide inhalation is chemical asphyxia, reducing the oxygen carrying power of the blood. The 100 ppm threshold limit can be interpreted from the results of extensive human experiment and examination of exposed workman. It will prevent injury, but will allow a recognizable effect if inhaled for eight hours.

Carbon tetrachloride. Elkins (1950, p. 229) on the basis of industrial experience, concluded the earlier figure of 100 ppm was too high and suggested 40 ppm. Adams, Spencer, Rowe, McCollister and Irish (1952) in extensive animal studies, found some effect on the liver in some species at all concentrations above 5 ppm. Smyth (1937-55) found rats survive eight hours at 3000 ppm, but 8000 ppm is fatal. Human anesthesia, or near anesthesia, is usually fatal from

kidney injury, while early narcosis occurs at a low concentration. Chronic toxicity is chiefly marked by liver injury.

The most important effect of carbon tetrachloride inhalation is chronic toxicity centering in the liver. The 25 ppm threshold limit can be interpreted from the results of repeated animal inhalations and industrial experience. It is low enough to prevent irreversible injury, but perhaps it will allow minor injury.

CELLOSOLVE *(2-ethoxyethanol)*. Werner, Nawrocki, Mitchell, Miller and von Oettingen (1943) found rats repeatedly inhaling 300 to 400 ppm showed small but measurable blood cell effects. Werner, Mitchell, Miller and von Oettingen (1943b) found dogs inhaling 800 ppm repeatedly developed small blood cell effects. Smyth (1937-55) found rats survive four hours at 2000 ppm, but half are killed by 4000 and all are killed by eight hours at 4000 ppm, close to saturation. Death is marked by severe kidney damage.

The most important effect of CELLOSOLVE inhalation is chronic poisoning centering in the red blood cells. The 200 ppm threshold limit can be interpreted from results of repeated animal inhalation studies. It appears to be low enough to prevent injury. There are no data to judge the degree of eye and nose irritation it allows.

CELLOSOLVE *acetate (2-ethoxyethyl acetate)*. Smyth (1937-55) found in dogs after 120 seven-hour inhalations of 600 ppm, only a small increase in bromosulfalein retention, with eye and nose irritation. Rats survive 1500 ppm (close to saturation) for four hours, but two of six die after eight hours. It is easily hydrolyzed to CELLOSOLVE and acetic acid. Systemic injury should follow closely that of CELLOSOLVE, but respiratory tract irritation is somewhat greater.

The most important effect of CELLOSOLVE acetate is chronic poisoning due to hydrolysis to CELLOSOLVE. The 100 ppm threshold limit can be interpreted from analogy with CELLOSOLVE. It appears low enough to prevent injury. There are no data to judge the degree of eye and nose irritation it allows.

CHLORDANE. Princi and Spurbeck (1951) quote animal data indicating effects are principally neurological, with liver and kidney injury and pulmonary irritation. They found workers for three years with exposures of the order of 5 mg./cu.m. showed no clinical evidence of effect. Alvarez and Hyman (1953) examined men in another producing plant with up to five years exposure and found no effects, but concentrations were not measured. Ingle (1953) shows that early reports of inhalation injury in animals were due to a volatile unreacted intermediate in the early product, and that 14 days continuous inhalation of saturated air does not injure mice. ACGIH (1954b) bases its tentative threshold limit on a rat oral LD_{50} of 590 mg./kg.

The most important effect of chlordane inhalation is chronic poisoning centering in the liver. The 2 mg./cu.m. threshold limit can be interpreted from the results of examinations of exposed workmen. It is low enough to prevent injury.

Chlorinated camphene, 60% (toxaphene). Lackey (1949) found an oral dose of 10 mg./kg. caused convulsions in dogs while 15 mg./kg. was fatal. A daily dose of 4 mg./kg. for 106 days was not fatal, but at times convulsions were seen. Liver and kidney changes resulted.

The most important effect of chlorinated camphene is chronic poisoning centering in the liver. The 0.5 mg./cu.m. tentative threshold limit can be interpreted from the results of single and repeated oral doses, and by analogy with the similar but less toxic DDT. It appears low enough to prevent injury.

Chlorinated diphenyl oxide. After extensive inhalation studies with rats, Drinker (1949) concluded that 0.5 mg./cu.m. is a permissible concentration which will not lead to systemic injury. Liver injury is the effect of chronic poisoning. Smyth (1937-55) found the material penetrates the skin, and repeated contact leads to chloracne.

The most important effect of chlorinated diphenyl oxide inhalation is chronic poisoning centering in the liver. The 0.5 mg./cu.m. threshold limit can be interpreted from the results of repeated animal inhalations. It is low enough to prevent injury.

Chlorine. Sklyanskaya and Rappaport (1935) found lung injuries and increased incidence of pneumonia in guinea pigs repeatedly inhaling 0.7 to 1.7 ppm. Fairhall (1950, p. 52) states it irritates eyes and nose, and may cause fatal lung irritation. Inhalation of 1000 ppm is rapidly fatal, 40

to 60 ppm may lead to pneumonitis and lung edema, 30 ppm causes coughing, 15 ppm throat irritation and 3.5 ppm can be smelled. Patty (1948-9, p. 547) concludes 1 to 2 ppm is tolerable, 3 to 6 ppm irritating.

The most important effect of inhalation of chlorine gas is respiratory tract irritation, with lung edema the maximum effect. The 1 ppm threshold limit can be interpreted from repeated animal inhalation and human sensory data. It is low enough to prevent injury.

Chlorine trifluoride. Horn and Weir (1955) found it an extremely active irritant. Rats are killed in 40 minutes at 96 ppm, while rats and dogs inhaling 5 ppm repeatedly are severely injured. Pneumonia was increased and there was respiratory difficulty in all. The vapors injure the cornea. Horn and Weir (1956) found repeated inhalation of 1.17 ppm by rats and dogs injured only by increased incidence of pneumonia.

The most important effect of inhalation of chlorine trifluoride gas is respiratory tract irritation, with lung edema the maximum effect. The 0.1 ppm threshold limit can be interpreted from repeated animal inhalation. It appears low enough to prevent injury.

Chlorobenzene. Fairhall (1949, p. 260) concludes it is somewhat more toxic than benzene, but finds no evidence of hematopoetic effect.

The most important effect of chlorobenzene inhalation is narcosis. The 75 ppm threshold limit can be interpreted only as a rough estimate. By comparison with other chlorinated hydrocarbons, it appears low enough to prevent injury.

Chlorobromomethane. Svirbely, Highman, Alford and von Oettingen (1947) found 3000 ppm to be the LC_{50} for mice in eight-hour inhalations. Exposures of rats, rabbits and dogs to 1000 ppm seven hours a day five days a week for fourteen weeks were without effect. Non-progressive liver injury was found from single inhalations, but liver and kidney remained normal during the repeated inhalations. Comstock et al. (1952) found light narcosis in rats and mice inhaling 3000 ppm for 10 to 15 minutes, and about 30,000 ppm was fatal within 15 minutes. Pulmonary edema was present in animals dying.

The most important effect of inhalation of chlorobromomethane is narcosis with nonprogressive effects on liver and kidney. The 400 ppm tentative threshold limit can be interpreted from the results of repeated inhalations with animals. It appears low enough to prevent injury.

Chlorodiphenyl (42% chlorine). After extensive inhalation studies with rats, Drinker (1939) concluded that 10 mg./cu.m. of a sample 68% chlorine, but free from chlorinated diphenyl benzene, would lead to no systemic injury, but that the presence of chlorinated diphenyl benzene reduced the permissible limit to 0.5 mg./cu.m. Smyth (1937-55) found the material penetrates the skin, and repeated contact leads to chloracne. Treon, Cleveland, Cappel and Atchley (1956) reported that inhalation of 8.6 mg./cu.m. for 24 seven-hour periods did not affect four species of animals, and 1.9 mg./cu.m. for 150 periods also was without effect.

The most important effect of chlorodiphenyl inhalation is chronic poisoning, centering in the liver. The 1 mg./cu.m. threshold limit can be interpreted from the results of repeated animal inhalations. It is low enough to prevent injury.

Chlorodiphenyl, 54% chlorine. Treon, Cleveland, Cappel and Atchley (1956) reported that repeated inhalation over a seven-month period of 1.5 mg./cu.m. caused some minor liver injury in four species of rodents.

The most important effect of inhalation of chlorodiphenyl (54% chlorine) is chronic toxicity centering in the liver. The 0.5 mg./cu.m. tentative threshold limit can be interpreted from the results of repeated inhalation by animals. It appears to be slightly below an injurious concentration.

Chloroform. Fairhall (1949, p. 264) concludes it acts much like carbon tetrachloride, and that anesthetic use has led to liver injury, but regression is more likely than with carbon tetrachloride. A concentration of 4000 ppm causes slight symptoms after several hours exposure. Patty (1948-9, p. 793) concludes the least concentration smelled is 200 to 300 ppm. Smyth (1937-55) found one of six rats die from four hours at 4000 ppm, and all from 8000 ppm.

The most important effect of inhalation of chloroform is chronic poisoning centering in the liver. The 100 ppm threshold limit can be interpreted from the results of single

animal inhalations and human sensory response. It appears low enough to prevent injury, but in view of changes in ideas about carbon tetrachloride, new data are desirable.

1-Chloro-1-nitropropane. Machle, Scott, Treon, Heyroth and Kitzmiller (1945) exposed animals for six hours to 400 ppm. Effects were chiefly irritation of eye, nose bronchi and lung, with some injury to liver, kidney and vascular system, and 25% died. Simultaneous repeated studies on 1,1-dichloro-1-nitroethane showed little cumulative action.

The most important effect of 1-chloro-1-nitroethane is lung injury. The 20 ppm threshold limit can be interpreted from the results of single animal inhalations. It appears low enough to prevent injury.

Chloropicrin. Fairhall (1949) concludes it is an irritant gas, producing bronchial and lung injury. He concludes 20 ppm produces lesions within one to two minutes and that 4 ppm will incapacitate a man.

The most important effect of chloropicrin is respiratory tract irritation with lung injury probable. The 1 ppm tentative threshold limit can be interpreted from summaries of studies of its use as a war gas. It appears low enough to prevent serious injury.

Chloroprene (2-chlorobutadiene). Von Oettingen, Hueper, Deichmann-Gruebler and Wiley (1936) made repeated inhalation studies with animals. A concentration of 83 ppm caused some respiratory irritation, central nervous system depression, and effects on liver, kidney and vascular system. Skin penetration was dangerous.

The most important effect of chloroprene inhalation is chronic poisoning, centering in the liver. The 25 ppm threshold limit can be interpreted from the results of repeated animal inhalations. It appears low enough to prevent injury.

Chromic acid. Bloomfield and Blum (1928) and Riley and Goldman (1937) demonstrated in industrial surveys that perforation of the nasal septum and other upper respiratory tract effects of chromic acid mists are not found at a concentration of about 0.1 mg./cu.m. Other effects are not produced. Vigliani and Zurlo (1955) found ulcerated nasal septum, inflamed larynx, chronic bronchitis and two respiratory tract cancers among 150 workers exposed to 0.11 to 0.15 mg./cu.m.

The most important effect of inhalation of chromic acid aerosol is upper respiratory tract irritation, leading to perforation of the nasal septum. The 0.1 mg./cu.m. threshold limit can be interpreted from studies of exposed workmen. It is low enough to prevent injury.

CRAG herbicide. ACGIH (1954b) suggests on the basis of a general low toxicity and a rat oral LD_{50} of 1500 mg./kg., that 10 mg./cu.m. is a fit limit. Smyth (1937-55) found death from a single oral dose due to respiratory paralysis with injuy to liver and kidney. Rats are not affected by 0.02% in their diet for two years, and 0.06% causes minor effects on liver and kidney.

The most important effect of CRAG herbicide inhalation is chronic poisoning centering in the liver. The 15 mg./cu.m. threshold limit can be interpreted from the results of single and repeated oral doses to animals. It appears low enough to prevent injury.

Cresol. Fairhall (1949, p. 271) concludes effects and practical hazards are like those of phenol. Smyth (1937-55) found rats survive eight hours inhalation of vapors substantially saturated at room temperature. The liquid penetrates the skin to a dangerous extent, and causes severe skin and corneal injury. The odor appears to be somewhat more intense than that of phenol.

Although cresol vapors are odorous and irritating, their major effect is chronic systemic poisoning. The 5 ppm threshold limit can be interpreted from analogy with phenol. It appears to be low enough to prevent chronic poisoning.

Cyanide as CN. To the extent that cyanide dusts dissolve, their toxicity is that of hydrogen cyanide, with some added local irritation due to hydrolysis on moist tissue. Cyanide dust equivalent of the 10 ppm threshold limit for hydrogen cyanide is 11 mg./cu.m. The 5 mg./cu.m. threshold limit is about half that for hydrogen cyanide, and hence is conservative.

Cyclohexane. Treon, Crutchfield and Kitzmiller (1943) found minor liver and kidney changes in animals repeatedly inhaling 786 ppm, none at 434 ppm. Fairhall (1949, p. 273) concludes acute poisoning is anesthesia and that repeated inhalation causes no hematopoetic changes. Patty

(1948-9, p. 769) reports 300 ppm has no distinct odor or irritation.

The most important effect of cyclohexane inhalation is narcosis, with non-progressive organic effects. The 400 ppm threshold limit can be interpreted from results of repeated animal inhalation. It is low enough to prevent definite narcosis.

Cyclohexanol. Treon, Crutchfield and Kitzmiller (1943) reported that repeated inhalation of 693 ppm caused minimal pathological changes in a monkey, and 145 ppm in the livers and kidneys of rabbits. Nelson, Ege, Ross, Woodman and Silverman (1943) found 100 ppm causes objectionable eye, nose and throat irritation in unacclimated subjects. Smyth (1937-55) was unable to kill rats by eight hours inhalation of substantially saturated vapors.

The most important effect of cyclohexanol inhalation is narcosis, with non-progressive organic effects less prominent. The 100 ppm threshold limit can be interpreted from results of repeated animal inhalations and human sensory data. It is low enough to prevent significant narcosis or injury, but not to prevent irritation.

Cyclohexanone. Treon, Crutchfield and Kitzmiller (1943) in animal experiments found only narcosis and irritation. Nelson, Ege, Ross, Woodman and Silverman (1943) found 50 ppm caused objectionable eye, nose and throat irritation in unacclimated subjects. Smyth (1937-55) did not kill rats by four hours at 4000 ppm, but 8000 ppm caused anesthetic death.

The most important effect of cyclohexanone inhalation is narcosis. The 100 ppm threshold limit can be interpreted from repeated animal inhalations and human sensory response. It is probably low enough to prevent definite narcosis.

Cyclohexene. Fairhall (1949, p. 279) concludes that 9000 ppm causes mild narcosis in animals, while 13,500 to 15,000 ppm gives anesthetic death. This indicates a toxicity greater than that of cyclohexane.

The most important effect of cyclohexene inhalation is narcosis. The 400 ppm threshold limit can be interpreted from results of single animal inhalations. It is low enough to prevent definite narcosis.

Cyclopropane. Fairhall (1949, p. 280) reviews reports of experience in surgical anesthesia and concludes there is no toxic hazard in industrial use. The most important effect of cyclopropane gas inhalation is narcosis. The 400 ppm threshold limit can be interpreted from analogy with cyclopentane, and by human surgical use. It is probably low enough to prevent definite narcosis.

2,4-D. Rowe and Hymas (1954) conclude that it has a low degree of chronicity, and the acute LD_{50} values range from 300 to 1000 mg./kg. for various species.

The most important effect of 2,4-D inhalation is chronic poisoning centering in the liver. The 10 mg./cu.m. threshold limit can be interpreted from the results of single and repeated oral doses to animals. It appears low enough to prevent injury.

DDT. Barnes (1953) finds no incidence of illness among workers using it throughout the world. Poisoning from accidental ingestion is marked by abdominal pain, vomiting, dizziness and weakness. Long repeated doses of 25 to 50 mg./kg. are required to poison animals, although man is probably more sensitive.

The most important effect of DDT inhalation is chronic poisoning centering in the liver. The 1 mg./cu.m. tentative threshold limit can be interpreted from the results of repeated oral doses to animals. It appears low enough to prevent injury.

Decaborane. Svirbely (1954a,b) found the LC_{50} for mice inhaling vapors for four hours to be 25.7 ppm. Symptoms included central nervous excitability and corneal opacity. Six-hour inhalations of 20 ppm by rats, repeated 20 times, killed some with fatty livers and central nervous excitability. Comstock and Oberts (1953) report that the median detectable odor is 0.35 mg./cu.m. (0.07 ppm), described as foul or chocolate-like.

The most important effect of decaborane inhalation is acute toxicity involving the central nervous system, with liver injury less important. The 0.05 ppm tentative threshold limit can be interpreted from limited animal inhalations. It appears to allow an adequate margin of safety.

Diacetone alcohol. Von Oettingen (1943, p. 138) reports animals at 2100 ppm are restless, irritation and kidney effects are noted. He concludes it is twice as toxic as acetone. Silverman, Schulte and First (1946) found 100 ppm irritating to eyes, nose and throat but not intolerable in un-

acclimated subjects. Smyth (1937-55) found 1500 ppm, approaching saturation, did not kill rats in eight hours.

The most important effect of diacetone alcohol inhalation is narcosis. The 50 ppm threshold limit can be interpreted from single animal inhalations and human response. It appears low enough to prevent definite narcosis.

Diborane. Rozendaal (1951) reported on five human injuries from inhalation of diborane and other boron hydrides. Diborane produced symptoms like metal fume fever and severe central nervous system irritation. Krachow (1953) reported that single inhalations of 50 ppm may be fatal to rats, resulting in lung injury, while 6 ppm repeatedly for three weeks causes lung damage, and 2 ppm causes some lung injury within four weeks. Kidney effects are also noted. He finds diborane about as injurious as phosgene. Odor is evident at 2 to 4 ppm.

The most important effects of diborane inhalation are central nervous system irritation and lung injury. The 0.1 ppm threshold limit can be interpreted from the effects of repeated animal inhalation and clinical studies on accidental human injuries. It is apparently low enough to prevent injury.

O-Dichlorobenzene. Cameron, Thomas, Ashmore, Warren, Buchan, and Kenny-Hughes (1937) found 30 minutes inhalation of 390 ppm caused in animals, liver necrosis and minor kidney injury. They concluded it is more toxic than chlorobenzene. Fairhall (1949, p. 284) points out its narcotic properties. Elkins (1950, p. 147) reports some irritation of eye and respiratory tract from 100 ppm, without other effects.

The most important effect of o-dichlorobenzene inhalation is chronic poisoning centering in the liver. The 50 ppm threshold limit can be interpreted from the results of single animal inhalations and human sensory response data. It does not appear to allow sufficient margin to prevent human injury from continuous inhalation.

Dichlorodifluoromethane. Sayers, Yant, Chornyak and Shoaf (1930) found animals exposed repeatedly to 200,000 ppm developed a generalized tremor and ataxic gait, but no gross pathology. Fairhall (1949, p. 347) notes it has little, if any anesthetic or toxic action.

The most important effect of dichlorodifluoromethane inhalation is asphyxia from extremely high concentrations. The 1000 ppm threshold limit can be interpreted from the results of repeated animal inhalations. It represents good engineering control rather than a hazard limit.

1,1-Dichloroethane. Henderson and Haggard (1943, p. 207) conclude it is similar to carbon tetrachloride. Smyth (1937-55) found rats survive eight hours at 4000 ppm, but are killed at 16,000 ppm, an acute toxicity half that of carbon tetrachloride. In repeated inhalations by rats and dogs, chronic toxicity somewhat less than that of carbon tetrachloride was likewise found.

The most important effect of 1,1-dichloroethane inhalation is chronic poisoning, centering in the liver. The 100 ppm threshold limit can be interpreted from single and repeated animal inhalations. It may be low enough to prevent injury, but new data are desirable in view of current views on carbon tetrachloride.

1,2-Dichloroethylene. Fairhall (1949, p. 292) concludes 39,000 to 50,000 ppm is lethal to guinea pigs, and 18,000 ppm produces narcosis. Acute poisoning consists of narcosis with central nervous system irritation. No liver injury has been found. The vapors are irritating. Smyth (1937-55) found the cis isomer did not kill nor anesthetize rats in four hours at 8000 ppm, while 16,000 ppm anesthetized in eight minutes and killed in four hours. The trans isomer was twice as toxic and anesthetic.

The most important effect of 1,2-dichloroethylene inhalation is narcosis. The 200 ppm threshold limit can be interpreted from the results of single animal inhalations. It is low enough to prevent definite narcosis.

Dichloroethyl ether. Schrenk, Patty and Yant (1933) found 500 to 1000 ppm killed guinea pigs in 30 to 60 minutes with lung hemorrhage and edema, while 35 ppm produced slight irritation in several hours. This concentration can be smelled but is not immediately irritating to man, while 500 to 1000 ppm is lacrimating. Smyth (1937-55) found rats survive four hours at 125 ppm, but are killed by 250 ppm. Skin penetration is moderately dangerous.

The most important effect of dichloroethyl ether inhalation is lung injury. The 15 ppm threshold limit can be interpreted from the results of single animal inhalations. It

seems to be low enough to prevent injury.

Dichloromonofluoromethane. Nuckolls (1933) showed it is little different from other fluorocarbons used as refrigerants. They are practically inert in the body.

The most important effect of dichloromonofluoromethane inhalation is asphyxia from extremely high concentrations. The 1000 ppm threshold limit can be interpreted by analogy with other fluorocarbon refrigerants. It represents good engineering control rather than a hazard limit.

1,1-Dichloro-1-nitroethane. Machle, Scott, Treon, Heyroth and Kitzmiller (1945) found 25 ppm did not kill animals in a total of 204 hours inhalation. The vapors irritated eyes, nose, bronchi and lungs, with injury to liver, kidney and vascular system.

The most important effect of 1,1-dichloro-1-nitroethane inhalation is lung injury. The 10 ppm threshold limit can be interpreted from the results of repeated animal inhalations. It appears low enough to prevent injury.

Dichlorotetrafluoroethane. Nuckolls (1933) and Yant, Schrenk and Patty (1932) found only transient discomfort in animals exposed two hours to 25,000 ppm. No chronic effects are to be expected from this physiologically inert material.

The most important effect of dichlorotetrafluorethane inhalation is asphyxia from very high concentrations. The 1000 ppm threshold limit can be interpreted from the results of single animal inhalations. It appears to be far below a possible injurious level.

Dieldrin. The workers examined by Princi and Spurbeck (1951) after three years industrial exposure to several related insecticides including dieldrin with concentrations of the order of 5 mg./cu.m., showed no evidence of clinical effect. Treon and Cleveland (1955) found 25 ppm in the diet of rats for two years did not shorten their lives. Dogs are most sensitive, tolerating only 3 ppm. Chronic toxicity centered in the liver. In acute poisoning, central nervous system irritation is prominent. Dieldrin penetrates the skin.

The most important effect of dieldrin inhalation is chronic poisoning centering in the liver. The 0.25 mg./cu.m. threshold limit can be interpreted from the results of repeated oral doses to animals and results of examination of exposed workmen. It is low enough to prevent injury.

Diethylamine. Brieger and Hodes (1951) exposed rabbits repeatedly to 50 ppm and found lung and corneal injury, but animals survived with some liver injury. Smyth (1937-55) found rats suffer fractional mortality from four hours at 2000 and 4000 ppm, while 8000 is lethal to all. The liquid is extremely injurious to the cornea.

The most important effect of diethylamine inhalation is respiratory tract irritation, with lung edema the maximum injury. The 25 ppm threshold limit can be interpreted from results of repeated animal inhalation. It appears low enough to prevent injury.

Difluorodibromomethane. ACGIH (1955b) quotes Chemical Corps Medical Laboratories Research Report No. 180, 1953. This shows six weeks daily inhalation of 2300 ppm kills some animals with lung injury, liver and central nervous system damage.

The most important effects of difluorodibromomethane inhalation are chronic toxicity and respiratory tract irritation. The 100 ppm threshold limit can be interpreted from the results of single animal inhalations, in comparison with carbon tetrachloride, ethyl and methyl bromide. It appears low enough to prevent injury.

Diisobutyl ketone. Silverman, Schulte and First (1946) found concentrations above 25 ppm give eye irritation in unacclimated subjects. Carpenter, Pozzani and Weil (1953) found single eight-hour inhalations of 200 ppm killed some rats by anesthesia, repeated inhalation of 1650 ppm killed some and caused liver, kidney and lung injuries, while 125 ppm was without effect. Two humans found 50 ppm satisfactory for work during a three-hour period, but were uncomfortable at 100 ppm.

The most important effect of diisobutyl ketone inhalation is narcosis. The 50 ppm threshold limit can be interpreted from repeated animal inhalations and human sensory response. It is low enough to prevent definite narcosis.

2,4-Diisocyanotoluene. Swenson, Holmquist and Lundgren (1955) quote French animal inhalations in which 1 to 2 gm./cu.m. (140 to 280 ppm) caused only respiratory tract irritation. They report three human industrial cases featuring upper respiratory tract irritation followed by sensitization

and asthma-like attacks. Unpublished American experience features sensitization.

The most important effect of 2,4-diisocyanotoluene inhalation is respiratory tract irritation, followed by sensitization. The 0.1 ppm tentative threshold limit cannot be interpreted quantitatively. It is probably not low enough to prevent an attack in a sensitized person.

Dimethyl aniline (N-dimethyl aniline). Henderson and Haggard (1943, p. 227) conclude that the alkyl anilines are less toxic than aniline, but von Oettingen (1941, p. 15) concludes dimethyl aniline has a greater depressant effect than aniline. It forms methemoglobin in the blood. He cites two human poisonings with symptoms like aniline.

The most important effect of dimethyl aniline inhalation is poisoning like that from aniline. The 5 ppm threshold limit can be interpreted by analogy with aniline. It appears low enough to prevent injury.

Dimethyl sulfate. Flury and Zernik (1931) report that 13 ppm severely poisoned cats in 20 minutes. Patty (1948-9, p. 925) states it has only a faint odor and there is a considerable latent period before effects are evident. Fairhall (1949, p. 309) concludes it is a powerful irritant upon inhalation, the liquid causes severe skin burns and corneal injury, and when swallowed, marked central nervous system effects such as convulsions and delirium result. Smyth (1937-55) found rats survive four hours inhalation of 15 ppm but die from 30 ppm.

The most important effect of dimethyl sulfate inhalation is delayed irritation of bronchi, and lung edema, not preceded by promptly evident irritation of eye and upper respiratory tract. Very high concentrations may cause convulsions and delirium, then coma. The 1 ppm threshold limit can be interpreted from results of single animal inhalations. It appears to be low enough to protect against lung injury, but available data do not indicate that it will prevent bronchial irritation.

Dinitrobenzene. Fairhall (1949) concludes the chief effect of dinitrobenzene is the production of methemoglobin, leading to anoxia and anemia. Von Oettingen (1941) in a review of the literature finds chronic liver injury and cites opinions that it is more toxic than nitrobenzene.

The most important effect of inhalation of dinitrobenzene is chronic poisoning. The 1 mg./cu.m. tentative threshold limit appears to be based on the reasonable assumption that dinitrobenzene is five times as toxic as nitrobenzene.

Dinitro-o-cresol. Baltimore (1943) reports a non-fatal case from inhalation of 4.7 mg./cu.m. Spencer, Rowe, Adams and Irish (1948) in animal experiments, found 10 to 50 mg./kg. is a fatal dose for animals. It is a rapidly acting metabolic stimulant, increasing body temperature to the point of heat stroke. Cataracts are produced in susceptible species, but chronicity is low.

The most important effect of dinitro-o-cresol inhalation is acute poisoning, marked by metabolic stimulation with rise of body temperature. The 0.2 mg./cu.m. threshold limit can be interpreted from the facts of one industrial accident. It appears low enough to prevent injury.

Dinitrotoluene. Von Oettingen (1941, p. 110) concludes this is similar to trinitrotoluene but less toxic when pure. The dust causes mucous membrane irritation.

The most important effect of dinitrotoluene inhalation is chronic poisoning, marked by central nervous system, liver and red blood cell changes. The 1.5 mg./cu.m. threshold limit can be interpreted from analogy with trinitrotoluene. It does not appear low enough to prevent all injuries.

Dioxane. Fairley, Linton and Ford-Moore (1934) found liver and kidney injury in animals repeatedly inhaling 1000 ppm, and from skin absorption. Silverman, Schulte and First (1946) found eye, nose and throat irritation at 300 ppm in unacclimated subjects. Patty (1948-9, p. 957) concludes there is only a faint odor at 200 ppm. Smyth (1937-55) found rabbits particularly susceptible, repeated inhalation at 800 ppm killing some with kidney injury within 30 days.

The most important effect of dioxane inhalation is chronic poisoning, centering in the liver and kidney. The 100 ppm threshold limit can be interpreted from results of repeated animal exposure studies. It appears to be low enough to prevent injury.

EPN. Hodge, Maynard et al (1954) found the acute oral LD_{50} for rats ranges from 7 to 33 mg./kg., while 75 ppm in the diet is without effect during two years. The ma-

terial is a cholinesterase inhibitor, and symptoms of excess in animals are confined to excitability and tremors. It appears to be ⅛ to ⅓ as toxic as parathion.

The most important effect of EPN inhalation is the reduction of blood cholinesterase. The 0.5 mg./cu.m. threshold limit can be interpreted from the results of single oral doses to animals and analogy with parathion. It appears low enough to prevent injury.

Ethyl acetate. The unacclimated subjects of Nelson, Ege, Ross, Woodman and Silverman (1943) found an objectionably strong odor at 200 ppm and eye, nose and throat irritation at 400 ppm. Henderson and Haggard (1943, p. 222) conclude 10,000 to 20,000 ppm is dangerous for short exposures. It is mildly narcotic but does not produce systemic effects. Smyth (1937-55) found rats inhaling 8000 ppm for four hours survive, but 16,000 ppm kills.

The most important effect of ethyl acetate inhalation is narcosis. The 400 ppm threshold limit can be interpreted by human sensory data and results of single inhalations by animals. It apparently is low enough to prevent definite narcosis.

Ethyl acrylate. Pozzani, Weil and Carpenter (1949) found 30 inhalations of 300 ppm injured lungs, liver and kidneys of rats and rabbits, while 70 ppm did not injure rats. They found that 8 ppm is readily detectable by odor, and that the odor of 50 ppm is objectionable. Treon, Sigmon, Wright and Kitzmiller (1949) obtained closely similar results.

The most important effect of ethyl acrylate inhalation is respiratory tract irritation. The 25 ppm tentative threshold limit can be interpreted from repeated animal inhalation results and limited human sensory data. It appears low enough to prevent injury.

Ethyl alcohol. Henderson and Haggard (1943, p. 219) consider its vapors anesthetic but not toxic. Only under exceptional circumstances can inhalation cause slight intoxication. They estimate 1064 ppm for eight hours is not sufficient to produce the earliest stage of intoxication in man. Cook (1945) stated that industrial exposure at 1000 ppm has led only to a few complaints of eye irritation. Patty (1948-9, p. 851) concludes 6000 to 9000 ppm is initially intolerably irritating, but that acclimatization follows. Smyth (1937-55) found rats survive eight hours at 16,000 ppm but some are killed at 32,000 ppm.

The most important effect of ethyl alcohol inhalation is narcosis. The 1000 ppm threshold limit can be interpreted from human physiological data. It is low enough to prevent significant narcosis, but some eye irritation will result.

Ethyl amine. Brieger and Hodes (1951) exposed rabbits repeatedly to 50 ppm and found lung and corneal injury, with some effect on heart muscle. Smyth (1937-55) found four hours at 4000 to 8000 ppm kills some rats, and 16,000 ppm kills all. Respiratory tract irritation is prominent. The liquid is extremely injurious to the cornea.

The most important effect of ethyl amine inhalation is respiratory tract irritation, with lung edema the maximum injury. The 25 ppm threshold limit can be interpreted from results of repeated animal inhalation. It appears low enough to prevent injury.

Ethyl benzene. Yant, Schrenk, Waite and Patty (1930) found only slight irritation in guinea pigs breathing 1000 ppm for eight hours. Fatalities from higher levels showed lung edema. Humans found 1000 ppm was initially irritating to the eyes and 2000 ppm caused throat irritation, constriction in the chest and slight intoxication. Smyth (1937-55) found rats not killed by four hours at 2000 ppm, while 8000 ppm is fatal. There is general agreement that it does not exert the hematopoetic effects of benzene.

The most important effect of ethyl benzene inhalation is narcosis, with irritation of the entire respiratory tract contributing to injury. The 200 ppm threshold limit can be interpreted from results of single animal inhalations and limited human sensory response. It is low enough to prevent injury.

Ethyl bromide. Waite and Yant (1928) found lung edema a prominent effect of guinea pig inhalation. Sayers, Yant, Thomas and Berger (1929) found several hours inhalation of 1700 ppm by animals was without effect. Henderson and Haggard (1943, p. 207) note that vapors are not narcotic.

The most important effect of ethyl bromide inhalation is respiratory tract irritation. The 200 ppm threshold limit can be interpreted from the results of single animal inhalations. It is probably low enough to prevent injury.

Ethyl chloride. Sayers, Yant, Thomas and Berger (1929) found with animals that 150,000 to 300,000 ppm is rapidly fatal, death being due to anesthesia, that 40,000 ppm does not kill in 270 minutes, 20,000 ppm causes only moderate unsteadiness, and 10,000 ppm is without effect.

The most important effect of ethyl chloride inhalation is narcosis. The 1000 ppm threshold limit can be interpreted from the results of single animal inhalations. It is low enough to prevent significant narcosis.

Ethylene chlorhydrin. Dierker and Brown (1944) reported one fatality with estimated exposure of 305 ppm for two hours. Animal experiment produced kidney pathology from 365 ppm for two hours. Goldblatt (1944) and Goldblatt and Chiesman (1944) report 11 cases, two of them fatal. Nervous system, cardiovascular system and kidneys were affected, and no warning irritation was apparent. They found one hour at 1120 ppm killed animals, with evidence of potential chronic effect. They conclude no concentration is safe for daily exposure. Smyth and Carpenter (1945) point out very rapid skin penetration of the liquid, the absence of warning skin irritation and the failure of rubber gloves to protect.

The most important effect of ethylene chlorhydrin inhalation is acute poisoning, centering in the kidney. The 5 ppm threshold limit can be interpreted from the results of single animal inhalations. It appears low enough to prevent injury.

Ethylene diamine. Dernehl (1951) related industrial experience showing this is a sensitizer upon contact and inhalation. Pozzani and Carpenter (1954) exposed rats repeatedly to the vapors. All died within 20 days at 484 ppm, with loss of hair, injury to the kidney and lesser effects on liver and lung. There was no effect except loss of hair at 132 ppm, and none whatever at 59 ppm. Very brief human exposures found 100 ppm inoffensive, tingling of skin and nose at 200 ppm and intolerable sensory response at 400 ppm. Smyth (1937-55) found eight hours at 2000 ppm did not kill rats while 4000 ppm was fatal. Death was due chiefly to kidney injury, with some injury to lung. The liquid irritates the skin and severely injures the cornea.

The most important effects of ethylene diamine inhalation are respiratory tract irritation, kidney damage and sensitization. The 10 ppm threshold limit can be interpreted from results of repeated animal inhalation and human sensory data. It is low enough to prevent irritation and injury, but probably not to eliminate response by persons already sensitized.

Ethylene dibromide. Rowe, Spencer, McCollister, Hollingsworth and Adams (1952) found four species of animals tolerated repeated inhalation of 25 ppm, but not 50 ppm. Major injury was in lung and liver, with kidney and central nervous system less prominent. The liquid penetrates the skin. It is painful in the eye, but causes only transient injury. The odor of a concentration dangerous to life is definite and sickening.

The most important effects of ethylene dibromide inhalation are respiratory tract irritation and liver injury. The 25 ppm threshold limit can be interpreted from the results of repeated animal inhalations. It is probably low enough to prevent injury.

Ethylene dichloride. Spencer, Rowe, Adams, McCollister and Irish (1951) studying repeated inhalation by animals, found no effect from 100 ppm. Single dangerous inhalations irritate the lung and depress the central nervous system, while dangerous repeated inhalations injure liver and kidney. They feel chronic intoxication is unlikely because tolerated repeated inhalations are close to concentrations tolerated once. Concentrations sufficient to cause marked narcosis are irritating to the upper respiratory tract. Adams, Spencer, Rowe, McCollister and Irish (1952) simultaneously studying carbon tetrachloride, found it at least four times as toxic as ethylene dichloride. Elkins (1950, p. 137) found complaints of nausea from industrial exposures to 100 to 150 ppm. Patty (1948-9, p. 805) finds little odor at 100 ppm, slight eye and nose irritation at 1000 ppm.

The most important effect of ethylene dichloride inhalation is chronic poisoning, centering in the liver. The 100 ppm threshold limit can be interpreted from the results of repeated animal inhalations and human response. It is low enough to prevent injury.

Ethylene imine. Silver and McGrath (1948) and Carpenter, Smyth and Shaffer (1948) studied single inhalations in animals. The LC_{50} for mice in a 10-minute exposure

is 2236 ppm, 500 ppm for one hour is fatal to rats and guinea pigs, 25 ppm kills some in eight hours, and 10 ppm kills none in eight hours. Symptoms and death are delayed, due to kidney tubular injury, with lesser lung and liver injury. Humans can barely smell 2 ppm, while 100 ppm begins to irritate eyes and nose. The liquid penetrates the skin, produces severe skin and corneal burns, and sensitizes the skin.

The most important effect of ethylene imine inhalation is acute poisoning, centering in the kidney, with lung injury of lesser importance. The 5 ppm threshold limit can be interpreted from results of single animal inhalation studies and limited human sensory data. It is apparently low enough to prevent poisoning and upper respiratory tract irritation.

Ethylene oxide. Waite, Patty and Yant (1930) in single animal inhalations found no symptoms from eight hours at 250 ppm. Greater concentrations caused eye and nose irritation, narcosis, bronchial and lung irritation. Sensory response is only moderate at low concentrations, but eye and nose irritation are intolerable at high concentrations. Sexton and Henson (1950) have called attention to spectacular human skin injuries with sensitization, which arise from contact with the liquid and its aqueous solutions. Smyth (1937-55) found rats survive four hours at 4000 ppm but are killed by 8000 ppm. Hollingsworth, Rowe, Oyen, McCollister and Spencer (1956) in animals repeatedly inhaling 204 ppm found lung irritation and some fatalities, with injury to liver, kidney, adrenal and testes. Rats and mice were not affected at 49 ppm, other species tolerated 113 ppm. Jacobson, Hackley and Feinsilver (1956) with dogs, rats and mice found some fatalities from repeated inhalation of 400 ppm, but 100 ppm had little affect.

The most important effect of ethylene oxide inhalation is respiratory tract irritation leading to lung injury, while injury to liver and kidney are less important. The 100 ppm threshold limit can be interpreted from results of repeated animal inhalation studies. It appears low enough to prevent injury.

Ethyl ether. Henderson and Haggard (1943, p. 195) estimate the maximum human blood concentration from inhalation of 400 ppm is 0.018 gm./l., causing no intoxication, while 2000 ppm will give a blood level of 0.09 gm./l., corresponding to instability in some persons. They state 35,000 ppm anesthetizes in 30 minutes, and a higher concentration kills by respiratory paralysis. Experience in human anesthesia shows that pneumonitis may follow ether anesthesia, but other injuries are unlikely. Nelson, Ege, Ross, Woodman and Silverman (1943) found nasal irritation at 200 ppm with unacclimated subjects, somewhat greater at 300 ppm.

The most important effect of ethyl ether inhalation is narcosis. The 400 ppm threshold limit can be interpreted from human physiological and sensory data. It is low enough to prevent definite narcosis.

Ethyl formate. Flury and Zernik (1931) reported 330 ppm causes in man slight eye irritation and rapidly increasing nasal irritation, while 10,000 ppm is anesthetic and fatal. Fairhall (1949, p. 344) notes its effects are irritation and narcosis, and that there is no chronic toxicity. Smyth (1937-55) found rats survive four hours inhalation of 4000 ppm but die from 8000 ppm.

The most important effect of ethyl formate inhalation is narcosis. The 100 ppm threshold limit can be interpreted from scanty human sensory data and single animal inhalations. It appears to be low enough to prevent definite narcosis and irritation.

Ethyl mercaptan. Sayers, Fieldner, Yant, Leitch and Pearce (1930) report the odor detectable at one part per billion and disagreeable at one part per fifteen million. They quote that its effects are like those of hydrogen sulfide, and by analogy with butyl mercaptan they conclude more than 733 ppm is required to kill in 30 minutes. Flury and Zernik (1931) quote 3000 ppm as harmless to dogs and 10,000 ppm causing hematologic and blood cell changes.

The most important effect of ethyl mercaptan is eye and respiratory tract irritation. The 250 ppm tentative threshold limit can be interpreted from the results of single inhalation by animals. It appears low enough to prevent injury and eye irritation.

Ethyl silicate. Smyth and Seaton (1940) found eight-hour inhalation of 550 ppm the least fatal exposure for guinea pigs. Death is due to lung injury, with some kidney

damage. Humans found 85 ppm detectable by odor, 250 ppm slightly irritating to eye and nose, and 3000 ppm extremely irritating. Rowe, Spencer and Bass (1948) found some kidney damage in rats repeatedly inhaling 125 ppm. Pozzani and Carpenter (1951) exposed rodents repeatedly. Some died within 30 days at 440 ppm with injury to lung, liver and kidney, but 88 ppm did not injure. The data suggest that repeated inhalation at a given concentration is no more injurious than a single inhalation.

The most important effect of ethyl silicate inhalation is lung injury, with non-progressive kidney damage less important. The 100 ppm threshold limit can be interpreted from results of repeated animal inhalations and limited human sensory data. It can be smelled, it is not irritating, and is low enough to prevent lung or kidney injury.

Ferbam. Hodge, Maynard, Downs, Blanchet and Jones (1952) reported the oral LD_{50} for rats to be over 17 gm./kg., with guinea pigs and rabbits more sensitive. Rats tolerated 0.01% in their diet for 30 days without effect while 0.5% was required to kill. Dogs were not injured by 25 mg./kg./day for six months. The mechanism of injury is not clear.

The most important effect of inhalation of Ferbam appears to be the upper respiratory tract irritation of a substantially inert dust. The 15 mg./cu.m. tentative threshold limit is in accord with this.

Ferro vanadium dust. Roshchin (1952) exposed rats two months to 1000 to 2000 mg./cu.m. and found no effect beyond some lung irritation. The author suggests a threshold limit of 1 mg./cu.m. Vanadium compounds irritate the upper respiratory tract, but Sjoberg (1951) found no chronic general poisoning in workers exposed to vanadium pentoxide dust, symptoms being confined to respiratory difficulties and skin allergies.

The most important effect of inhalation of ferrovanadium dust is respiratory tract irritation. The 1 mg./cu.m. threshold limit can be interpreted from limited repeated animal inhalations and examination of exposed workmen. It appears low enough to prevent injury.

Fluoride dust. Roholm (1937) found fluorosis of human bone, but no other effects, after several years work in 2 to 3 ppm fluorine, equivalent to 1.5 to 2.3 mg./cu.m. soluble fluoride dust. Higher concentrations provide respiratory tract irritation and effects on liver and kidney. Largent (1952) found storage in the body occurs when as little as three milligrams per day fluoride in the form of sodium fluoride is ingested, roughly equivalent to inhalation of 0.3 mg./cu.m. soluble fluoride dust.

The most important effect of inhalation of fluoride dust is chronic poisoning, centering in the bones, with respiratory tract irritation at high concentrations. The 2.5 mg./cu.m. threshold limit can be interpreted from results of examination of exposed workmen and experimental studies of human fluoride retention. It is not low enough to prevent fluoride storage with resulting effects on the bones.

Fluorine. Machle and Evans (1940) examined workmen exposed intermittently to as much as 10 ppm and found no clinical evidence of damage, but there was some accumulation in bones and teeth. Stokinger (1949) found few toxic effects in dogs repeatedly exposed to 0.5 ppm. Greater concentrations injured lung and kidney. This is apparently below the level which leads to bone abnormalities (Roholm 1937).

The most important effect of inhalation of fluorine gas is respiratory tract irritation, with lung edema the maximum effect. Chronic effect on bone metabolism is also important. The 0.1 ppm threshold limit can be interpreted from results of animal inhalation and studies on exposed workmen. It is low enough to prevent injury.

Fluoroacetates. Dieke and Richter (1946) report the median lethal oral dose to be 0.22 mg./kg. for wild rats. The substance is rapidly fatal through intervention in the tricarboxylic acid metabolic cycle.

The most important effect of fluoroacetate inhalation is acute toxicity. The 0.1 mg./cu.m. tentative threshold limit can be interpreted from acute oral toxicity data for rats. It corresponds to a maximum human intake of one milligram per day, apparently well below a dangerous amount.

Fluorotrichloromethane. Nuckolls (1933) found in animals no more than occasional tremors and retching during two hours at 22,000 to 25,000 ppm. No toxic effects are to be expected from this physiologically inert material.

The most important effect of fluorotrichloromethane inhalation is a minor degree of narcosis, and asphyxia from very high concentrations. The 1000 ppm threshold limit can be interpreted from the results of single animal inhalations. It appears to be far below a possibly injurious level.

Formaldehyde. Henderson and Haggard (1943, p. 128) conclude its action is chiefly irritation of all tissues contacted, particularly the respiratory tract, and that systemic effects are not important. Skin sensitization to solutions is frequent and respiratory tract sensitization to the gas is not unlikely. Elkins (1950, p. 231) reports irritation in workmen inhaling 5 to 6 ppm, and eye irritation of unhardened persons at lower levels. Smyth (1937-55) found rats survive eight hours inhalation of 125 ppm but are killed by 250 ppm. The liquid causes severe corneal injury.

The most important effect of formaldehyde inhalation is irritation, first evident in the eyes, then in the upper respiratory tract, bronchi and even lung. The 5 ppm threshold limit can be interpreted from uncontrolled human sensory data. It is sufficiently low to prevent lung injury.

Furfural. Fairhall (1949, p. 354) quotes animal experiments in which inhalation of 280 ppm resulted only in slight mucous membrane irritation, while 2800 ppm caused acute irritation, prostration and lung edema. ACGIH (1954b) quotes Korenman and Resnik, (*Arch. Hyg.*, 104:344, 1931) to the effect that 2 to 14 ppm causes human headache and eye irritation. Severe corneal injury is to be expected from the fluid, and analogy with other aldehydes suggests that skin and respiratory sensitization may be found.

The most important effect of furfural inhalation seems to be irritation, first evident in the eyes, then in the upper respiratory tract, bronchi and even lung. The 5 ppm tentative threshold limit can be interpreted from uncontrolled human sensory data. It is sufficiently low to prevent lung injury.

Furfuryl alcohol. Fine and Wills (1950) found that death from furfuryl alcohol is due to the respiratory paralysis of anesthesia, and that short of death, its effects are reversible. ACGIH (1955b) quotes Chemical Corps Medical Laboratories Research Report No. 139, 1942, to the effect that eight hours inhalation of 700 ppm killed 25% of a group of rats.

The most important effect of furfuryl alcohol inhalation is narcosis. The 50 ppm tentative threshold limit can be interpreted from results of single animal inhalations. It appears low enough to prevent injury.

Gasoline. Sayers, Fieldner, Yant and Thomas (1927) reported human dizziness at 700 ppm, and Drinker, Yaglou and Warren (1943) found human respiratory tract irritation and headache begin at 1000 ppm. Elkins (1950, p. 99) found industrially no sensory response to 660 to 800 ppm benzine, and dizziness at 2000 to 3000 ppm. He concludes chronic effects do not occur when concentrations are too low to cause narcosis, but Hayhurst (1936) reported chronic poisoning consisting of central nervous system effects and blood cell changes after many years of exposure. This article does not seem to be generally accepted. Henderson and Haggard (1943, p. 192) consider nausea and incoordination the significant effects, with anesthetic death at 20,000 to 30,000 ppm. Aromatic hydrocarbons in gasoline from cracking operations may much reduce safety.

The most important effect of gasoline inhalation is narcosis. The threshold limit of 500 ppm can be interpreted from human sensory data, both experimental and industrially observed. It is low enough to prevent definite narcosis.

Heptane. Patty and Yant (1929) reported slight human dizziness from 1000 ppm. The paraffin hydrocarbons are anesthetic agents and irritate mucous membrane, but they do not cause systemic toxicity.

The most important effect of heptane inhalation is narcosis. The 500 ppm threshold limit can be interpreted from limited human sensory data and by analogy with the better studied gasoline. It is probably low enough to prevent definite narcosis.

HETP (hexaethyltetraphosphate). This material is substantially identical in quantitative and qualitative effect with the cholinesterase inhibitor TEPP. Apparently no data specifically upon inhalation have been published.

The most important effect of HETP is reduction of blood cholinesterase. The 0.1 mg./cu.m. tentative threshold limit can be interpreted by analogy with parathion. It

appears to be consistent with that value.

Hexane. Nelson, Ege, Ross, Woodman and Silverman (1943) with unacclimated subjects found no irritation at 500 ppm. Drinker, Yaglou and Warren (1943) found human nausea, headache, eye and throat irritation at 1400 to 1500 ppm. The paraffin hydrocarbons anesthetize and irritate mucous membrane, but do not cause systemic toxicity.

The most important effect of hexane inhalation is narcosis. The 500 ppm threshold limit can be interpreted from human sensory data. It is low enough to prevent definite narcosis.

Hexanone (methyl butyl ketone). Simple ketones are irritant and narcotic, but not chronically toxic. Schrenk, Yant and Patty (1936) found guinea pigs tolerate 1000 ppm with slight or no symptoms for several hours, a concentration with a strong odor, moderately irritating to human eyes and nose. Death from higher levels is anesthetic respiratory paralysis. Specht, Miller, Valaer and Sayers (1940) found it more depressant than acetone, butanone or pentanone. Smyth (1937-55) found rats survive four hours at 4000 ppm, but die at 8000 ppm.

The most important effect of hexanone inhalation is narcosis. The 100 ppm threshold limit can be interpreted from the results of single animal inhalations and limited human response data. It is low enough to prevent definite narcosis.

Hexone (methyl isobutyl ketone). Simple ketones are irritant and narcotic, but not chronically toxic. Specht (1938) and Specht, Miller, Valaer and Sayers (1940) found guinea pigs are not injured by several hours at 1000 ppm, but men find eye and nose irritation. Smyth (1937-55) found rats survive four hours at 2000 ppm, but die from 4000 ppm. Silverman, Schulte and First (1946) found the odor objectionable at 200 ppm, eye irritation evident, but no nose and throat irritation with unacclimated subjects.

The most important effect of hexone inhalation is narcosis. The 100 ppm threshold limit can be interpreted from results of single animal inhalations and human sensory response. It is low enough to prevent definite narcosis.

Hydrazine. Comstock, Lawson, Greene and Oberst (1954) found in animals damage to lung and liver, tremors and convulsions, irritation of eye, nose and throat. Most of the animals repeatedly inhaling 20 ppm died by 30th day. During six month's inhalation of 5 ppm, only minor changes in the lungs of rats and dogs were found.

The most important effect of inhalation of hydrazine is respiratory tract irritation. The 1 ppm threshold limit can be interpreted from results of repeated animal inhalation. It is probably low enough to prevent injury.

Hydrogen Bromide. ACGIH (1955b) quotes unpublished human response data from the Connecticut Bureau of Industrial Hygiene. Odor was evident at 2 ppm, nose and throat irritation began to be evident at 3 ppm, and eye irritation was not evident at 6 ppm.

The most important effect of hydrogen bromide inhalation is respiratory tract irritation, with lung edema the maximum effect. The 5 ppm tentative threshold limit can be interpreted from analogy with hydrogen chloride and human sensory response. It is probably low enough to prevent injury.

Hydrogen chloride. Machle, Kitzmiller, Scott and Treon (1942) found animals not affected by repeated inhalation of 34 ppm. Henderson and Haggard (1943, p. 126) consider it an irritant without systemic effect. They state 1000 to 2000 ppm is dangerous to life through lung edema in a short time. Elkins (1950, p. 79) finds 10 ppm highly irritating to humans although immunity seems to develop; 5 ppm immediately irritating; and even lower levels can erode the teeth.

The most important effect of hydrogen chloride inhalation is respiratory tract irritation, with lung edema the maximum effect. The 5 ppm threshold limit can be interpreted from repeated animal inhalation and human sensory data. It is low enough to prevent injury.

Hydrogen cyanide. Flury and Zernik (1931) give 19 to 36 ppm as tolerable for six hours without symptoms. Henderson and Haggard (1943, p. 173) conclude injury is chemical asphyxia, and chronic toxicity is not to be expected. They state 3000 ppm is rapidly fatal, 100 to 240 ppm dangerous in 30 to 60 minutes, and 20 to 40 ppm gives slight symptoms in several hours. This is one of the few gases which can penetrate the skin in dangerous amounts. Patty (1948-9, p. 633) finds the odor barely detectable at 0.9 ppm, and recognizable at 2 to 5 ppm.

The most important effect of hydrogen cyanide inhalation is acute poisoning, a chemical asphyxia. The 10 ppm threshold limit can be interpreted from results of older single animal inhalation data and some human sensory data. It is low enough to prevent injury.

Hydrogen fluoride. Stokinger (1949) found 30 ppm is highly toxic to animals, causing pulmonary damage, kidney and testis changes and increases in bone fluoride. Animals tolerated 7 ppm with only mild respiratory tract irritation in repeated exposure. Elkins (1950, p. 73) reports nosebleeds at 0.4 to 0.7 ppm. Patty (1948-9, p. 543) finds 0.026 mg./l. (22 ppm) is slowly irritating and at 0.1 mg./l. (120 ppm) the skin smarts. The liquid causes severe slowly healing skin injuries, and destroys the cornea. All soluble fluorides interfere with calcium metabolism and an excess produces bone and tooth abnormalities.

The most important effect of hydrogen fluoride inhalation is respiratory tract irritation, with lung edema the maximum effect. Chronic effect on bone metabolism is also important. The 3 ppm threshold limit can be interpreted from results of repeated animal inhalation and human sensory data. It is low enough to prevent injury

Hydrogen peroxide, 90%. Oberst, Comstock and Hackley (1954) found rats survive eight hours at 250 to 300 ppm without symptoms, but irritation and areas of edema are found in the lungs. Dogs survived six months at 7 ppm without injury, although the skin was thickened and the lungs irritated. The liquid is extremely destructive to skin and cornea.

The most important effect of hydrogen peroxide aerosol inhalation is respiratory tract irritation, with lung edema the maximum effect. The 1 ppm threshold limit can be interpreted from results of repeated animal inhalation. It is low enough to prevent injury.

Hydrogen selenide. Dudley and Miller (1941) found animals are killed in eight hours at 0.3 to 1.2 ppm, with lung irritation and changes in liver and spleen. Eye and nose irritation made 1.5 ppm intolerable to man, but 0.3 ppm is not irritating and perception of its odor is soon lost. Buchan (1947) reported industrial cases due to less than 0.2 ppm, with liver injury.

The most important effect of hydrogen selenide is acute poisoning, largely lung edema at high concentration, and chronic poisoning centering in the liver. The 0.05 ppm threshold limit can be interpreted from the results of single animal inhalations and industrial accidents. It appears low enough to prevent injury

Hydrogen sulfide. Henderson and Haggard (1943, p. 140, 243) state hydrogen sulfide may cause very rapid death from respiratory paralysis, or delayed death from lung injury. It is not cumulative. Low concentrations irritate the cornea. A concentration fatal in 30 minutes is 600 ppm, while 70 to 150 ppm causes slight symptoms in several hours. Barthelemy (1939) found no injury among viscose workers during 10 years, with control at about 20 ppm. Elkins (1950, p. 232) found eye irritation in industry at 10 ppm and some complaints even at 5 ppm. Patty (1948-9, p. 590) concludes 0.3 ppm can be smelled, 3 to 5 ppm is offensive.

The most important effect of hydrogen sulfide inhalation is acute toxicity, marked by respiratory paralysis or lung edema. The 20 ppm threshold limit can be interpreted from the results of examination of exposed workmen. It is low enough to prevent injury.

Hydroquinone. Sterner, Oglesby and Anderson (1947) reported on several years industrial experience with men exposed to quinone vapor and hydroquinone dust. No systemic effects could be found, but high concentrations caused transient eye irritation, and after several years, a pigmentation of cornea and conjunctiva was apparent, due to local action on the exposed tissue. Loss of vision has followed pigmentation in some cases, according to Oglesby (1956). It is uncertain whether the vapor or the dust was responsible. Hydroquinone dust ranged from 0.12 to 13 mg./cu.m. After comparing exposure with concentration, the authors conclude hydroquinone dust should be kept below 2 to 3 mg./cu.m.

The most important effect of hydroquinone inhalation is transient eye irritation and a slowly developing pigmentation in the eye. Visual disability can result. The 2 mg./cu.m. threshold limit can be interpreted from the results of examination of exposed workmen. It appears low enough to prevent effect.

Iodine. Henderson and Haggard (1943, p. 133) state it is a respiratory tract irritant with more effect on the lungs than chlorine and bromine. They quote an 1889 thesis by Matt to the effect that 0.1 ppm does not disturb workers. Fairhall (1949, p. 90) points out that excessive absorption can disturb the metabolism through effect on the thyroid.

The most important effect of iodine vapor inhalation is respiratory tract irritation, with lung edema the maximum injury. The 0.1 ppm threshold limit can be interpreted from analogy with chlorine. It appears low enough to prevent injury.

Iron oxide fume. Fairhall (1949, p. 92) reviews several articles. Inhalation of iron oxide dust for 5 to 10 years can lead to a benign pneumoconiosis, siderosis, revealed by x-ray but not injurious. U.S. Department of Labor (1941) in studies of welders, concluded iron oxide fume below 30 mg./cu.m. was without effect, while in excess of this level a chronic bronchitis may result.

The most important effect of inhalation of iron oxide fume is bronchitis or metal fume fever. The 15 mg./cu.m. threshold limit can be interpreted by analogy with zinc oxide fume. It is low enough to prevent injury.

Isophorone. Smyth, Seaton and Fischer (1942) found no effect upon animals from repeated inhalation of 25 ppm, while 50 ppm caused some lung and kidney injury. Single inhalation of 4600 ppm (saturation) for eight hours injured the lung but did not anesthetize or kill. Silverman, Schulte and First (1946) found odor objectionable at 10 ppm and eye, nose and throat irritation at 25 ppm with unacclimated subjects.

The most important effects of isophorone inhalation are lung and kidney injury. The 25 ppm threshold limit can be interpreted from the results of repeated animal inhalation and human sensory data. It seems low enough to prevent injury.

Isopropylamine. Smyth, Carpenter and Weil (1951) reported that rats survive four hours inhalation of 4000 ppm, but die from 8000 ppm. This is half the acute toxicity Smyth (1937-55) found for butyl amine. ACGIH (1955b) cite unpublished industrial experience that levels above 5 ppm tend to be irritating.

The most important effect of isopropylamine inhalation is respiratory tract irritation, with lung edema the maximum injury. The 5 ppm threshold limit can be interpreted from analogy with ethyl amines. It is probably low enough to prevent injury.

Lead. Russell, Jones, Bloomfield, Britten and Thompson (1933) after a survey of a storage battery plant, proposed that the threshold limit be reduced from 0.5 to 0.15 mg./cu.m. At this level disabling lead poisoning does not occur, and mild poisoning is rare.

The most important effect of lead dust inhalation is chronic poisoning. The 0.15 mg./cu.m. threshold limit can be interpreted from the results of examinations of exposed workmen. It is low enough to prevent disabling poisoning, but not to prevent some mild cases.

Lead arsenate. Fairhall and Miller (1941) and Fairhall, Miller and Weaver (1943) found the arsenate in lead arsenate decreases lead absorption or increases lead excretion, and reduces lead storage.

The most important effect of inhalation of lead arsenate is chronic arsenic poisoning. The 0.15 mg./cu.m. tentative threshold limit can be interpreted from the results of biochemical studies on animals. It appears to have been selected by analogy with the limit for lead, while analogy with arsenic would yield a much higher figure.

Lindane. A.M.A. (1951) in a summary, states lindane in large doses acts as a central nervous system stimulant, leading to hyperirritability, convulsions and death. Animals exposed several months to saturated vapors were not affected, but 10,000 mg./cu.m. dust for one hour killed one of two mice. ACGIH (1954b) quotes an unpublished 1951 report by J. F. Treon et al. A year of repeated inhalation of 0.7 mg./cu.m. caused minimal pathology in animals. An unpublished 1952 thesis by Spear is quoted to the effect that 655 days at 0.19 mg./cu.m. 24 hours a day did not result in pathology in rats.

The most important effect of lindane inhalation is chronic poisoning centering in the liver. The 0.5 mg./cu.m. threshold limit can be interpreted from the results of repeated animal inhalations. It appears low enough to prevent injury.

Magnesium oxide fume. Drinker, Thomson and Finn (1927) reported that experi-

mental fume fever in man results from excessive inhalation, but does not occur below a concentration of 15 mg./cu.m. This condition is transient fever with chills, muscular pain, nausea and vomiting. An immunity is apparently build up.

The most important effect of inhalation of magnesium oxide fume is transient metal fume fever. The 15 mg./cu.m. threshold limit can be interpreted from the results of extensive human experiment. It is low enough to prevent injury.

Malathon. Johnson, Fletcher, Nolan and Cassaday (1952) reviewed toxicity data and conclude malathon is about one-hundredth as toxic to mammals as parathion. Tousey (1954) reviews data and confirms that its toxicity is considerably lower than that of many other cholinesterase inhibitors. Culver, Caplan and Batchelor (1955) found a group of entomologists with maximum exposure about five hours at a peak of 56 mg./cu.m. and an average of about 3.3 mg./cu.m. This had no effect on blood cholinesterase.

The most important effect of malathon inhalation is the reduction of blood cholinesterase. The 15 mg./cu.m. threshold limit can be interpreted from the results of oral doses to animals and limited examinations of exposed workmen. It appears low enough to prevent injury.

Manganese. Flinn, Neal and Fulton (1951) describe poisoning as an effect upon the basal brain ganglia, leading to disability from weakness in the legs, spastic gait, stolid mask-like expression and emotional disturbances, but not ordinarily shortening life. In an ore-crushing plant, they found no symptoms in men exposed to 30 mg./cu.m. or less and they concluded concentrations can be effectively limited to 6 mg./cu.m.

The most important effect of inhalation of manganese dust is chronic poisoning. The 6 mg./cu.m. threshold limit can be interpreted from the results of examination of exposed workmen. It is low enough to prevent injury.

Mercury. Neal et al. (1941) in a study of the felt hat industry, found the incidence of mercurialism proportional to atmospheric concentrations, with no cases found below 0.1 mg./cu.m. Chronic symptoms consist of psychic disturbances, timidity, tremors, pallor, salivation and tenderness of the gums. Ashe, Largent, Dutra, Hubbard and Blackstone (1953) in repeated inhalations by animals, found no effects at 0.1 mg./cu.m., but damage to kidney and brain at 0.86 mg./cu.m.

The most important effect of inhalation of mercury is chronic poisoning. The 0.1 mg./cu.m. threshold limit can be interpreted from the results of repeated animal inhalation and examination of exposed workmen.

Mercury (organic compounds). Ahlmark (1948) on the basis of Swedish industrial experience suggests a limit of 0.01 mg./cu.m. Lundgren and Swensson (1949) consider concentrations fluctuate so widely that analysis does not detect important peaks, and that an M.A.C. cannot be defined. Organo mercurials produce effects like mercury, but they have the added hazard of ready penetration of the skin. Trakhtenberg, (abstracted from the Russian in *Chemical Abstracts* 44:10162g, 1950) reported mice die at 10 to 30 mg./cu.m. within three to five hours and concludes 0.01 mg./cu.m. should not be tolerated for repeated human exposures.

The most important effect of inhalation of organo mercurials is chronic mercury poisoning. The 0.01 mg./cu.m. threshold limit can be interpreted from the results of industrial experience and single animal inhalations. It is probably low enough to prevent injury.

Mesityl oxide. Smyth, Seaton and Fischer (1942) found no effect upon animals from repeated inhalation of 50 ppm, while higher concentrations killed by anesthesia, with minor lung, kidney and liver injuries. In single inhalations, 100 ppm did not injure in eight hours, 500 killed some and 2,500 killed all, and in one hour 13,000 ppm (saturation) was fatal by anesthesia. Little cumulative action was revealed. Silverman, Schulte and First (1946) found some eye irritation at 25 ppm, and at 50 ppm nose irritation and a persistent unpleasant taste in unacclimated subjects.

The most important effect of mesityl oxide inhalation is narcosis. The 50 ppm threshold limit can be interpreted from the results of repeated animal inhalations and human sensory response. It is low enough to prevent definite narcosis.

Methoxychlor. Haag, Finnegan, Larson, Riese and Dreyfuss (1950) found methoxy-

chlor acts similarly to DDT in animals, and is less toxic by inhalation. Hodge, Maynard and Blanchet (1952) found no effect on rats in two years at 0.020% in the diet and no mortality or histological changes at 0.16%. Little accumulates in body fat.

The most important effect of methoxychlor inhalation is chronic poisoning centering in the liver. The 15 mg./cu.m. threshold limit can be interpreted from the results of repeated oral doses to animals. It appears low enough to prevent injury.

Methyl acetate. Fairhall (1949, p. 375) notes irritation of eye and respiratory tract, narcosis less prominent than from higher acetates, but fatal dose close to anesthetic dose, and symptoms persistent after appreciable narcosis. Death is due to anesthesia, but lung injury also occurs. Smyth (1937-55) found rats survive four-hour inhalations of 16,000 ppm, but die from 32,000 ppm.

The most important effect of methyl acetate inhalation is narcosis. The 200 ppm threshold limit can be interpreted by analogy with ethyl acetate, allowing a margin for greater irritation and for the slow metabolism of methyl alcohol. It apparently is low enough to prevent narcotic symptoms.

Methyl Acetylene. ACGIH (1955b) cites Horn, Chemical Corps Medical Laboratories Contract Report #35, 1954. For six months dogs and rats inhaled 28,700 ppm repeatedly. A few died. There was lung irritation and some central nervous system excitation.

The most important effect of methyl acetylene inhalation is lung injury. The 1000 ppm threshold limit can be interpreted from results of repeated animal inhalations. It is low enough to prevent injury.

Methyl acrylate. Treon, Sigmon, Wright and Kitzmiller (1949) exposed animals repeatedly to vapor. They found that 130 seven-hour inhalations of 31 ppm had no effect upon four species, except some loss in weight. Higher concentrations caused respiratory tract irritation and some narcosis.

The most important effect of methyl acrylate inhalation is respiratory tract irritation. The 10 ppm tentative threshold limit can be interpreted from repeated animal inhalations. It appears to be low enough to prevent injury.

Methylal (dimethoxymethane). Weaver, Hough, Highman and Fairhall (1951) found that high concentrations produce fatty changes in liver, kidney and heart of animals, with lung irritation. The threshold for chronic effects is 11,300 ppm.

The most important effect of methylal inhalation is chronic poisoning, centering in liver and kidneys, with narcosis and lung injury less important. The 1000 ppm threshold limit can be interpreted from results of repeated animal inhalation studies. It appears low enough to prevent injury, but there are no data to judge the degree of irritation and narcosis it allows.

Methyl alcohol. Sayers, Yant, Schrenk, Chornyak, Pearce, Patty and Linn (1942) found no effect on dogs from repeated inhalation of 450 to 500 ppm. Henderson and Haggard (1943, p. 218) stress slow elimination, leading to progressive rise in blood level from daily inhalation. At 200 ppm 0.87 grams can be absorbed by a human in eight hours, but only part of this can be eliminated before the next day. Methanol is primarily a narcotic agent, but it may injure retina and optic nerve, leading to cloudy vision or blindness. There is some irritation of mucous membranes. Elkins (1950, p. 111) found industrial exposures ranging from 100 to 1700 ppm with no evidence of poisoning. Smyth (1937-55) found rats survive eight-hour inhalations of 32,000 ppm, and only a fraction are killed by 64,000 ppm.

The most important effect of methyl alcohol inhalation is narcosis, with injury to retina and optic nerve likely only from quite excessive inhalation. The 200 ppm threshold limit can be interpreted from results of repeated animal inhalations. It will not cause significant narcosis, but continuous inhalation will cause a daily rise in the degree of early narcosis, due to slow elimination.

Methyl bromide. Irish, Adams, Spencer and Rowe (1940) found no effect from repeated inhalation at 17 ppm, and 34 ppm injured only rabbits. Watrous (1942) found mild symptoms in one-third of 90 workers in concentrations generally under 35 ppm. Ingram (1951) found injuries where workers were exposed to 100 to 1000 ppm. After improvements reduced exposure to about 20 ppm, injuries ceased. Fairhall (1949, p. 376) notes it is a respiratory tract irritant, a liver injurant, and a central nervous system poison leading to delirium, convulsions and even mania. It is rapidly metabolized and eliminated.

The most important effect of methyl bromide inhalation is chronic poisoning, centering in the central nervous system and liver. The 20 ppm threshold limit can be interpreted from the results of repeated animal inhalations and examination of exposed workmen. It appears to be rather precisely set at the maximum concentration humans tolerate without effect.

Methyl CELLOSOLVE (methoxyethanol). Donley (1936) and Parsons and Parsons (1938) reported toxic encephalopathy with granulopenic anemia from industrial exposure to a mixed solvent containing methyl CELLOSOLVE. Greenberg, Mayers, Goldwater, Burke and Moskowitz (1938) estimated concentrations in the establishment some time after the cases developed, and found 25 ppm of the glycol ether. Werner, Mitchell, Miller and von Oettingen (1943, a,b) in experiments on dogs did not confirm the degree of toxicity suggested by the human cases. The blood cell effects were obtained from repeated inhalation of 500 ppm, but no encephalopathy was found. Fairhall (1949, p. 336) concludes the vapors are somewhat irritating, and produce narcosis and kidney changes. Smyth (1937-55) found rats survive four hours at 2000 ppm, but die from eight hours.

The most important effect of methyl CELLOSOLVE inhalation is chronic poisoning, centering in the brain and red blood cells. The 25 ppm threshold limit can be interpreted from atmospheric analyses of doubtful validity after human industrial injuries, and from the results of repeated animal inhalations. It appears lower than is required to prevent injuries.

Methyl CELLOSOLVE acetate (2-methoxyethyl acetate.) This ester hydrolyzes in the body to methyl CELLOSOLVE and acetic acid, and its vapors are somewhat more irritating than those of the former.

The most important effect of methyl CELLOSOLVE acetate inhalation is chronic poisoning due to hydrolysis to methyl CELLOSOLVE. The 25 ppm threshold limit can be interpreted from analogy with methyl CELLOSOLVE. It is low enough to prevent injury.

Methyl chloride. Sayers, Yant, Thomas and Berger (1929) found 10 hours at 400 ppm produce no serious injuries in guinea pigs. Smith and von Oettingen (1947) found repeated inhalation of 300 ppm by six species has no effect, but 500 ppm causes central nervous system effects. Complete recovery from injury is slow. Fairhall (1949, p. 379) concludes it acts chiefly by narcosis, but liver, kidney and bone marrow injuries are found.

The most important effect of methyl chloride inhalation is central nervous system injury, with less important chronic poisoning. The 100 ppm threshold limit can be interpreted from the results of repeated animal inhalations. It is low enough to prevent injury.

Methyl chloroform. Adams, Spencer, Rowe and Irish (1950) found in repeated exposures that 650 ppm retarded guinea pig growth, and 3000 ppm caused slight liver effects. Three other species were less sensitive. Rats were mildly narcosed in one hour at 5000 ppm. The level tolerated in repeated inhalation was close to that tolerated in a single exposure. They concluded it is close to methylene chloride in toxicity and less toxic than trichloroethylene.

The most important effect of methyl chloroform inhalation is narcosis. The 500 ppm threshold limit can be interpreted from the results of repeated animal inhalation. It is low enough to prevent definite narcosis.

Methyl cyclohexane. Treon, Crutchfield and Kitzmiller (1943) found no effect on rabbits exposed 300 hours to 1162 ppm, slight kidney and liver changes from 90 hours at 2886 ppm and fractional mortality, eye and entire respiratory tract irritation and narcosis at 7308 ppm. Patty (1948-9, p. 770) concludes the odor is weak at 500-800 ppm.

The most important effect of methylcyclohexane inhalation is narcosis, with non-progressive organic changes. The 500 ppm threshold limit can be interpreted from results of repeated animal inhalation. It is low enough to prevent definite narcosis.

Methyl cyclohexanol. Treon, Crutchfield and Kitzmiller (1943) found 300 hours at 121 ppm causes slight liver and kidney changes in rabbits, and 300 hours at 503 ppm causes eye irritation with some narcosis, but does not kill. Patty (1948-9, p. 881) concludes that odor and irritation are evident at 500 ppm.

The most important effect of methyl cyclohexanol inhalation is narcosis, with non-progressive organic effects less promi-

nent. The 100 ppm threshold limit can be interpreted from results of repeated animal inhalation. It is low enough to prevent significant narcosis, and probably to prevent minor effects on liver and kidneys.

Methyl cyclohexanone. Treon, Crutchfield and Kitzmiller (1943) found no effect on rabbits from 300 hours at 182 ppm, slight eye irritation at 514 ppm, while 1822 ppm caused some narcosis and eye irritation.

The most important effect of methyl cyclohexanone inhalation is narcosis. The 100 ppm threshold limit can be interpreted from results of repeated animal inhalations. It appears low enough to prevent definite narcosis.

Methylene chloride. Heppel, Neal, Perrin, Orr and Porterfield (1944) found repeated inhalation of 10,000 ppm caused moderate narcosis in animals, some deaths from lung edema and liver damage. A concentration of 5000 ppm for six months had no effect upon four species, but reduced the voluntary activity of rats, indicative of a very early stage of narcosis not usually detected in animals. Fairhall (1949, p. 296) mentions a human fatality after accidental anesthesia.

The most important effect of inhalation of methylene chloride is chronic poisoning centering in the liver. The 500 ppm threshold limit can be interpreted from the results of repeated animal inhalations. It is low enough to prevent injury.

Methyl formate. Schrenk, Yant, Chornyak and Patty (1936) found guinea pigs tolerate 1500 to 2000 ppm for several hours without disturbance, and 5000 ppm for one hour. Symptoms of higher concentrations were nose and eye irritation, lung irritation, narcosis and anesthetic death.

The most important effect of methyl formate inhalation is narcosis. The 100 ppm threshold limit can be interpreted from results of single inhalations by animals. It appears to be low enough to prevent definite narcosis.

Methyl isobutyl carbinol (methyl amyl alcohol). Silverman, Schulte and First (1946) at 50 ppm found eye irritation in unacclimated subjects, although the odor was not objectionable. Experience with other alcohols indicates systemic effects are not to be expected. Smyth (1937-55) found rats inhaling 1000 ppm for eight hours are not killed, but 2000 ppm is fatal.

The most important effect of methyl isobutyl carbinol inhalation is narcosis. The 25 ppm threshold limit can be interpreted from human sensory data. It is low enough to prevent significant narcosis and irritation.

Methyl mercaptan. ACGIH (1954b) quotes de Rikowski who in 1893 found methyl mercaptan similar to but somewhat less toxic than hydrogen sulfide.

The most important effect of methyl mercaptan is eye and respiratory tract irritation. The 50 ppm tentative threshold limit can be interpreted by analogy with hydrogen sulfide. It appears low enough to prevent injury and eye irritation.

Molybdenum. Fairhall, Dunn, Sharpless and Pritchard (1945) in animal experiments, found some bronchial and alveolar irritation, with fatty changes in liver and kidneys. Animals survived repeated exposure one hour daily to 53 mg./cu.m. molybdic oxide fume and only one of a group was killed by 286 mg./cu.m. molybdenite dust. The Industrial Hygiene Digest (16:1083, 1952) abstracts Mogilevskaya to the effect that histopathological changes in rat heart, liver and kidney are found after repeated inhalation of 3 to 10 mg./cu.m. molybdenum oxide aerosol.

The most important effect of inhalation of molybdenum compounds is chronic poisoning, centering in liver and kidney. The threshold limits of 5 mg./cu.m. for soluble and 15 mg./cu.m. for insoluble molybdenum compounds can be interpreted from the results of repeated animal inhalations. They appear low enough to prevent injury, but slight toxic effects may result from soluble compounds.

Naphtha (coal tar). A mixture of toluene and xylene chiefly. The predominantly narcotic effects of these closely similar materials are additive and a mixture is no more injurious than the sum of its components. Cook (1945) points out that if a sample has a low boiling point, an appreciable content of benzene is to be suspected, and working concentrations should be reduced accordingly.

Being composed chiefly of toluene and xylene, the most important effect of coal tar naphtha inhalation, when free from benzene, is narcosis with irritation of the respiratory tract less important. The 200 ppm

threshold limit can be interpreted by analogy with those of toluene and xylene. It is low enough to prevent injury.

Naphtha (petroleum). A mixture of paraffin hydrocarbons of somewhat higher molecular weight than gasoline. The discussion under gasoline applies.

Nickel carbonyl. Fairhall (1949, p. 114) quotes Armit to the effect that this produces a deposit of finely divided nickel in the respiratory tract, leading to irritation and lung edema. Hueper (1950) summarizes the inferences that it produces cancer, originating in the nasal sinuses. Kincaid, Strong and Sunderman (1953) found 30 minutes at 10 ppm killed mice, 270 ppm killed cats, and they suggest toxicity may be related to body weight, with man surviving high concentrations. They found lung edema and severe liver injury, but conclude it is not cumulative and that a tolerance develops. Sunderman and Kincaid (1954) report on 36 human cases, two fatal. The fatalities were delayed, due to lung edema.

The immediate effect of nickel carbonyl vapor inhalation is lung irritation and delayed edema. Cancer originating in the nasal sinuses has been reported from longtime exposure. The 0.001 ppm threshold limit is apparently an approximation of zero, designed to prevent cancer. It is low enough to prevent all possibility of immediate effect, but there are no data to judge its effectiveness in preventing cancer.

Nicotine. Wilson and De Eds (1936) fed diets containing nicotine to growing rats for a 60-day period. Rats did not survive on 0.05% nicotine. Rats were not affected by 0.006% nicotine, equivalent to 4 mg./kg. body weight per day. A greater concentration reduced growth, due largely but not entirely to reduced food intake. Lehman (1949) estimates the fatal human dose to be 60 milligrams.

The most important effect of nicotine inhalation is ill-defined chronic poisoning. The 0.5 mg./cu.m. tentative threshold limit can be interpreted from repeated feeding studies on rats. It corresponds to a maximum human intake of five milligrams per day, apparently well below an injurious level.

Nitric acid. Fairhall (1949, p. 81) concludes it is an upper respiratory tract irritant, injuring the bronchi and even the lungs in high concentration. It also erodes the teeth. He suggests a threshold limit of 10 ppm.

The most important effect of inhalation of nitric acid is upper respiratory tract irritation. The 10 ppm tentative threshold limit appears to be based upon undocumented analogy with other acid gases.

p-Nitroaniline. Fairhall (1949, p. 402) concludes p-nitroaniline is more toxic than aniline, causing headache, nausea and cyanosis. This is based on British industrial experience.

The most important effect of p-nitroaniline inhalation is acute poisoning. The 1 ppm threshold limit is apparently an estimate based on analogy with aniline. It appears low enough to prevent injury.

Nitrobenzene. Henderson and Haggard (1943, p. 227) note that injury by skin absorption is more frequent than by inhalation. The compound is an anesthetic, producing methemoglobin and reducing blood pressure. Acute toxicity is marked by headache, narcosis, cyanosis, and death from respiratory paralysis. Chronic absorption results in anemia, cyanosis, muscular weakness, bladder irritation. The maximum concentration which gives no serious disturbance in one hour is 200 ppm, and 40 to 80 ppm cause symptoms in several hours.

The most important effect of nitrobenzene inhalation is chronic poisoning, marked by reduced blood pressure and cyanosis. The 1 ppm threshold limit can be interpreted from results of single animal inhalation. It appears low enough to prevent injury.

Nitroethane. Machle, Scott and Treon (1940) found guinea pigs inhaling 500 ppm for a total of 140 hours are not injured, but some die from 1000 ppm. Eye and nose irritation, narcosis, central nervous system irritation and lung edema are produced. Toxic symptoms are evident before narcosis.

The most important effect of nitroethane inhalation is acute poisoning accompanied by narcosis and irritation. The 100 ppm threshold limit can be interpreted from results of animal inhalation. It appears low enough to prevent injury.

Nitrogen dioxide. Henderson and Haggard (1943, p. 137) state that 62 ppm causes immediate throat irritation, 300 ppm coughing and 100 to 150 ppm is danger-

ous for 30 to 60 minutes. Fairhall (1949, p. 117) finds the vapor irritates the entire respiratory tract, leading to delayed lung edema. There is some reduction in blood pressure, causing headache. Gray, McNamee and Goldberg (1952) found respiratory tract inflammation in rats, inhaling 9 ppm for a total of 48 hours in 10 days. Patty (1948-9, p. 610) reports 5 ppm is evident by odor, 10 to 20 ppm is irritating to eyes and nose. Vigliani and Zurlo (1955) in workers exposed several years to 30 to 35 ppm found no symptoms. They regard a threshold limit of 15 ppm satisfactory when ozone is absent.

The most important effect of nitrogen dioxide inhalation is respiratory tract irritation, with delayed lung edema probable. The 5 ppm threshold limit can be interpreted from results of repeated animal inhalation and human sensory data. It appears low enough to prevent injury.

Nitroglycerine. Cook (1945) quotes U. S. Public Health Service experience of no systemic effects from 10 ppm, but with as little as 0.5 ppm causing severe headache upon return to work after a week-end. Fairhall (1949, p. 405) notes headache from lowered blood pressure, excitement, dizziness, fainting, cyanosis, death from respiratory paralysis. Acclimatization is prominent, skin penetration is a major hazard. Elkins (1950, p. 160) found some headaches at 0.04 ppm.

The most important effect of nitroglycerine inhalation is acute poisoning, marked by reduced blood pressure. The 0.5 ppm threshold limit can be interpreted from studies on exposed workmen. It is sufficiently low to prevent injury, but not to prevent headache.

Nitromethane. Machle, Scott and Treon (1940) found animals not affected by 500 ppm for a total of 140 hours, but 1000 ppm was fatal. The vapors are eye and respiratory irritants and mildly narcotic. Central nervous system irritation and lung edema result. Toxic symptoms are evident before narcosis.

The most important effect of nitromethane inhalation is acute poisoning, accompanied by narcosis and irritation. The 100 ppm threshold limit can be interpreted from results of repeated animal inhalation. It appears low enough to prevent injury.

2-Nitropropane. The animal work of Machle, Scott and Treon (1940) concluded that in the nitro paraffins toxicity increases with molecular weight. Thus, 2-nitropropane should be more toxic than nitroethane. Skinner (1947) found workmen in concentrations of 10 to 30 ppm were not affected, but 20 to 45 ppm caused anorexia, nausea, vomiting and diarrhea.

The most important effect of 2-nitropropane inhalation is acute poisoning accompanied by narcosis and irritation. The 50 ppm threshold limit can be interpreted from results of repeated animal inhalation and industrial experience. It is probably low enough to prevent injury, but not to prevent disturbing symptoms.

Nitrotoluene. Von Oettingen (1941) reviewed the literature and could find no clear distinction between the toxicities of nitrotoluene and nitrobenzene.

The most important effect of nitrotoluene inhalation is chronic poisoning, marked by reduced blood pressure and cyanosis. The 5 ppm threshold limit appears to be an estimate without quantitative support. It is doubtful whether the difference between nitrobenzene and nitrotoluene is sufficient to justify the difference in threshold limits.

Octane. No useful published data specifically upon octane were found, but analogy with heptane and gasoline is close.

The most important effect of octane inhalation is narcosis. The 500 ppm threshold limit can be interpreted only by analogy with pentane and gasoline. It is probably low enough to prevent definite narcosis.

Ozone. Fairhall (1949, p. 122) quotes McDonnell's incompletely reported work to the effect that daily inhalation of 0.1 ppm killed guinea pigs with pneumonia, higher concentrations leading to lung edema. He notes respiratory tract irritation with fatal pneumonitis or lung edema, but no systemic poisoning. He quotes a statement that 0.015 ppm can be smelled, and any higher concentration is irritating.

The most important effect of ozone inhalation is respiratory tract irritation, with lung edema the maximum effect. The 0.1 ppm threshold limit can be interpreted from results of limited repeated animal inhalations and human sensory data. It appears low enough to prevent injury.

Parathion. The earliest effect of this cholinesterase inhibitor is a reduction of

that enzyme activity in the blood. Brown and Bush (1950) in tests on industrial workers found inhalation of 0.1 to 0.8 mg./cu.m. causes definite reduction in blood cholinesterase activity. Vigliani and Zurlo (1955) on the basis of cholinesterase reduction conclude that a threshold limit of 0.07 to 0.12 mg./cu.m. is satisfactory.

The most important effect of parathion inhalation is the reduction of blood cholinesterase. The 0.1 mg./cu.m. threshold limit can be interpreted from the results of examination of exposed workmen. It is not certain that it is low enough to prevent measurable reduction of blood cholinesterase, but it is low enough to prevent injury.

Pentaborane. Svirbely (1954a,b) found the LC_{50} for mice inhaling vapors for two hours to be 10.9 ppm. Symptoms were central nervous excitability and corneal opacity. Six-hour inhalations of 3.3 ppm by rats killed all within four repetitions. Comstock and Oberst (1953) report that the median detectable odor is 2.5 mg./cu.m. (0.5 ppm), described as garlic or slightly sweet.

The most important effect of pentaborane inhalation is acute toxicity involving the central nervous system. The 0.01 ppm tentative threshold limit can be interpreted from limited repeated inhalations by animals. It appears to be a conservative estimate.

Pentachlornaphthalene. After extensive inhalation studies with rats, Drinker (1939) concluded that injury is exclusively in the liver and that the permissible limit for repeated inhalation is 0.5 mg./cu.m. The solid material or its oil solutions penetrates the skin, and repeated contact leads to chloracne.

The most important effect of pentachlornaphthalene inhalation is chronic poisoning centering in the liver. The 0.5 mg./cu.m. threshold limit can be interpreted from the results of repeated animal inhalations. It is low enough to prevent injury.

Pentachlorophenol. Kehoe, Deichmann-Gruebler and Kitzmiller (1939) found no evidence of chronic poisoning in rabbits, and the smallest lethal intravenous dose was 22 mg./kg. It penetrates the skin readily. Internal injury is primarily to the vascular system with heart failure.

The most important effect of pentachlorophenol inhalation is acute poisoning centering in the circulatory system. The 0.5 mg./cu.m. threshold limit can be interpreted from the results of repeated doses to animals. It appears low enough to prevent injury.

Pentane. Patty and Yant (1929) found no effect on humans from 10 minutes inhalation of 5000 ppm. Fairhall (1949, p. 358) concludes that only narcosis and irritation are produced.

The most important effect of pentane inhalation is narcosis. The 1000 ppm threshold limit can be interpreted from limited human sensory data and analogy with the better studied gasoline. It is probably low enough to prevent definite narcosis.

Pentanone (methyl propyl ketone). Yant, Patty and Schrenk (1936) found 30,000 to 50,000 ppm killed guinea pigs in 30 to 60 minutes; 1500 ppm caused slight or no symptoms in several hours and was strongly odorous and irritating to human eye and nose. Death was anesthetic. Smyth (1937-55) found four hours at 2000 ppm killed part of a group of rats.

The most important effect of pentanone inhalation is narcosis. The 200 ppm threshold limit can be interpreted from the results of single animal inhalation and limited human response data. It is low enough to prevent definite narcosis.

Perchloroethylene. Carpenter (1937) found rats inhaling 230 ppm for 150 days showed slight non-progressive liver and kidney effects, while 70 ppm had no effect. Humans perceived the odor at 50 ppm, slight eye irritation at 500 ppm, light narcosis at 1000 ppm, nausea at 5000 ppm. Rowe, McCollister, Spencer, Adams and Irish (1952) found repeated inhalation of 400 ppm does not affect rats, rabbits and monkeys, while 100 ppm does not affect guinea pigs. Humans found no symptoms at 100 ppm, minimum narcosis at 200 ppm, eye and nose irritation at 600 ppm, painful irritation at 1000 ppm.

The most important effect of perchloroethylene inhalation is narcosis. The 200 ppm threshold limit can be interpreted from the results of repeated animal inhalations and human response. It appears low enough to prevent significant narcosis.

Perchloromethyl mercaptan. ACGIH (1954b) cites Flury and Zernik (1931) to the effect that after 15 minutes inhalation of

45 ppm, mice and cats die within two days.

The most important effect of perchloromethyl mercaptan inhalation is eye and respiratory tract irritation. The 0.1 ppm tentative threshold limit can be interpreted on the basis of limited single inhalations by animals. It appears to be low enough to prevent injury.

Phenol. Deichmann, Kitzmiller and Witherup (1944) found guinea pigs are severely injured by 20 days inhalation of 25 to 50 ppm, rabbits suffer lung injury in 63 days, but rats are not affected. They conclude human injury from repeated inhalation is marked by digestive disturbance, nervous disorders, skin eruption and liver and kidney damage. The liquid penetrates the skin to a dangerous extent, and causes severe skin and corneal injury. Patty (1948-9, p. 1034) reports that 5 ppm can be recognized by odor. Smyth (1937-55) found rats survive eight hours inhalation of vapors saturated at room temperature.

Although phenol vapors are odorous and irritating, their major effect is chronic systemic poisoning. The 5 ppm threshold limit can be interpreted from results of repeated inhalations by animals. It appears to be low enough to prevent chronic toxic effects.

Phenylhydrazine. Von Oettingen (1941, p. 158) concludes death from a large dose is due to respiratory paralysis. There is hemolytic anemia, formation of methemoglobin, injury to the liver and heart muscle. Skin penetration is rapid and dermal sensitization takes place. The fatal oral dose for rats is of the order of 0.04 gm./kg.

The most important effect of phenylhydrazine inhalation is chronic poisoning, centering in the red blood cells. The 5 ppm threshold limit can be interpreted from analogy with aniline. Quantitative data are not available to judge the effectiveness of the limit.

Phosgene. Fieldner, Katz and Kinne (1921) cite the Chemical Warfare Service as authority for a 1 ppm allowable concentration for prolonged exposure, based on human tests. Henderson and Haggard (1943, p. 137) consider phosgene a lung injurant producing delayed edema. They say 3.1 ppm is immediately irritating to throat, 4 ppm to eyes, 4.9 ppm causes coughing, 5.6 ppm detectable by odor and 50 ppm rapidly fatal.

The most important effect of phosgene inhalation is respiratory tract irritation with delayed lung edema probable. The 1 ppm threshold limit can be interpreted from results of human studies. It is low enough to prevent injury.

Phosphine. Henderson and Haggard (1943, p. 243) report 1000 to 2000 ppm fatal in 30 minutes, 100 to 200 ppm the maximum for one hour without serious disturbance. Fairhall (1949, p. 127) quotes Müller to the effect that animals die from two four-hour inhalations of 20 ppm, or 7 of 10 ppm, but two months at 5 ppm did not injure. Acute poisoning is rapidly fatal with convulsions, paralysis and coma. Chronic poisoning is marked by anemia and nervous disturbances. Patty (1948-9, p. 576) concludes there is only a faint odor at 1 ppm.

The most important effect of phosphine inhalation is chronic poisoning. The 0.05 ppm threshold limit can be interpreted from the results of repeated animal inhalation. It appears low enough to prevent injury.

Phosphorous (yellow). Fairhall (1949, p. 131) summarizes the literature. The effects of chronic poisoning are upon bone metabolism. They may be noticed first as painful swollen gums, or as spontaneous fractures of the long bones. They develop into periostitis and necrosis of the lower jaw, with secondary infection. In single exposures one milligram per kilogram is usually fatal.

The most important effect of phosphorous inhalation is chronic poisoning, centering in the bones. The 0.1 mg./cu.m. threshold limit cannot be interpreted in quantitative terms.

Phosphorous pentachloride. Henderson and Haggard (1943, p. 134) conclude this material is an irritant to nose, throat and lungs through hydrolysis to hydrogen chloride. Skin burns from the solid are likely. Mice are killed in 10 minutes by 120 ppm.

The most important effect of phosphorous pentachloride inhalation is respiratory tract irritation, with lung edema possible. The 1 mg./cu.m. threshold limit is apparently an estimate without quantitative support. It appears low enough to prevent injury.

Phosphorous pentasulfide. Fairhall (1949, p. 131) quotes Barillet to the effect that phosphorous pentasulfide is somewhat less hazardous than phosphorous pentachloride.

The most important effect of phosphorous pentasulfide inhalation is respiratory tract

irritation. The 1 mg./cu.m. threshold limit is apparently an estimate without quantitative support. It appears low enough to prevent injury.

Phosphorous trichloride. Henderson and Haggard (1943, p. 134) consider it an irritant and lung injurant. They cite 600 ppm as rapidly fatal, and 2 to 4 ppm as the maximum for 30 to 60 minutes without serious disturbance. Cook (1945) quotes Butjog as finding 0.7 ppm causes only slight irritation in animals.

The most important effect of phosphorous trichloride inhalation is respiratory tract irritation, with lung edema the maximum effect. The 0.5 ppm threshold limit can be interpreted from limited animal inhalation data. It appears low enough to prevent injury.

Picric acid. Fairhall (1949, p. 423) describes systemic poisoning as gastroenteritis, hemorrhagic nephritis and hepatitis. Sunderman, Weidman and Batson (1945) studied workers handling ammonium picrate in atmospheres from 0.0088 to 0.1942 mg./cu.m. They found little respiratory tract irritation, no systemic effects but considerable dermatitis.

The most important effect of picric acid inhalation is chronic poisoning. The 0.1 mg./cu.m. threshold limit can be interpreted from observations on exposed workmen. It is low enough to prevent systemic injury but not respiratory tract irritation and sensitization.

Propyl acetate. Fairhall (1949, p. 426) concludes it is more irritating than ethyl acetate, more narcotic than ethyl or methyl acetates, but less lethal. Some respiratory tract irritation and liver injury are found. Death is due to anesthesia, but even deep narcosis may leave no after effects. Smyth (1937-55) found four hours inhalation of 32,000 ppm kills four of six rats.

The most important effect of propyl acetate inhalation is narcosis. The 200 ppm threshold limit can be interpreted by analogy with ethyl acetate, not by data. It appears to be low enough to prevent definite narcosis.

Propyl alcohol, iso. Nelson, Ege, Ross, Woodman and Silverman (1943) found 400 ppm causes mild irritation of eye, nose and throat, and 800 ppm is no more severe in unacclimated subjects. Fairhall (1949, p. 429) concludes it is similar to ethyl alcohol with no delayed effects, but twice as toxic. Smyth (1937-55) found rats survive four hours at 12,000 ppm, but half are killed in eight hours.

The most important effect of isopropyl alcohol inhalation is narcosis. The 400 ppm threshold limit can be interpreted from human sensory data and analogy with ethyl alcohol. It is low enough to prevent significant narcosis, but some irritation will result.

Propylene dichloride. Heppel, Neal, Highman and Porterfield (1946) found repeated inhalation of 1000 ppm by animals killed the first animal in seven days with severe liver effects. They conclude it is more toxic than ethylene dichloride but less so than carbon tetrachloride.

The most important effect of propylene dichloride inhalation is chronic poisoning centering in the liver. The 75 ppm threshold limit can be interpreted from the results of repeated animal inhalations. It appears low enough to prevent injury.

Propylene imine. Carpenter, Smyth and Shaffer (1948) found rats and guinea pigs killed by four hours at 500 ppm, and not by 30 minutes. This is about ⅛th the acute toxicity of ethylene imine in simultaneous work.

The most important effect of propylene imine vapor inhalation is acute poisoning, centering in the kidney, with lung injury of lesser importance. The 25 ppm threshold limit can be interpreted from results of scanty single animal inhalation studies and analogy with ethylene imine. It is apparently low enough to prevent injury.

Propyl ether (isopropyl ether). Machle, Scott and Treon (1939) found incomplete anesthesia in animals at 30,000 ppm, light narcosis at 10,000 ppm, and no effect in repeated exposures at 1000 ppm. They conclude it is 1.5 to 2 times as active as ethyl ether and less so than gasoline.

The most important effect of isopropyl ether inhalation is narcosis. The 500 ppm threshold limit can be interpreted from results of repeated animal inhalation. In comparison with data on ethyl ether it does not seem low enough to prevent definite narcosis.

Pyrethrum. There is little published on the toxicity of pyrethrum but many years of wide use as an insecticide indicates a low

degree of hazard, except for some slight skin sensitizing property. Carpenter, Weil, Pozzani and Smyth (1950) found the rat oral LD_{50} of two samples to be 0.82 and 1.87 gm./kg. Inhalation studies were made with an insecticidal aerosol containing 10% pyrethrum and 90% peanut oil and a Freon propellant. Rats inhaled a concentration of 6000 mg./cu.m. pyrethrum with 10 times as much peanut oil for 30 minutes, and only moderate lung congestion resulted. Rats and dogs inhaled a concentration of 16 mg./cu.m. pyrethrum with 10 times as much peanut oil for 40 thirty-minute periods during 31 calendar days, without injuries greater than those in peanut oil controls. Lehman (1949) estimates the fatal human dose to be 100 grams.

The most important effect of pyrethrum inhalation is irritation of the upper respiratory tract. The 2 mg./cu.m. tentative threshold limit can be interpreted from limited repeated animal inhalation data. It appears low enough to prevent injury.

Pyridine. Pollock, Finkelman and Arieff (1943) using pyridine for human therapy, found no toxic symptoms after daily doses of 0.31 to 1.54 ml., but 1.85 to 2.46 ml. was toxic, with one death of liver and kidney injury. Fairhall (1949, p. 434) considers small repeated doses affect the bone marrow, increasing the platelet count. Elkins (1950, p. 167) quotes a Czech report of mild central nervous symptoms at 6 to 12 ppm.

The most important effect of pyridine inhalation is chronic poisoning, centering in liver, kidney and bone marrow. The 10 ppm threshold limit can be interpreted from limited human symptom data. Mild symptoms may be found.

Quinone. Sterner, Oglesby and Anderson (1947) reported on several years industrial experience with men exposed to quinone vapor and hydroquinone dust. No systemic effects could be found, but high concentration caused transient eye irritation, and after several years a pigmentation of cornea and conjunctiva was apparent, due to local action on the exposed tissue. Loss of vision has followed pigmentation in some cases, according to Oglesby (1956). It is uncertain whether the vapor or the dust was responsible. Concentrations from 0.01 to 3.2 ppm quinone were found in the plant. The odor of quinone is perceptible at about 0.1 ppm, definite at about 0.15 ppm, irritating at 0.5 ppm and markedly irritating at 3 ppm. After comparing exposure with concentration, the authors conclude quinone vapor should be kept below 0.1 ppm.

The most important effect of quinone inhalation is transient eye irritation and a slowly developing pigmentation in the eye. The 0.1 ppm threshold limit can be interpreted from the results of examination of exposed workmen. It appears low enough to prevent effect.

Rotenone. On the basis of the literature and his own work, Lehman (1949) estimates the fatal human dose to be 200 grams by mouth.

The most important effect of rotenone inhalation is irritation of the upper respiratory tract. The 5 mg./cu.m. tentative threshold limit can be interpreted by analogy with pyrethrum. It appears low enough to prevent injury.

Selenium compounds (as Se). Fairhall (1949, p. 145) summarizes experimental studies which stress oral doses of selenium dioxide and inhalation of hydrogen selenide. Even at 3 ppm dioxide in the diet rats are injured, while 10 ppm kills within eight weeks. Dudley and Miller (1941) found animals killed in eight hours at 0.3 to 1.2 ppm hydrogen selenide, primarily due to lung injury, with changes in liver and spleen. Buchan (1947) reports industrial cases due to less than 0.2 ppm (0.65 mg./cu.m.) hydrogen selenide, with symptoms largely referable to the liver.

The most important effect of inhalation of selenium-bearing dusts is chronic poisoning, centering in the liver. The 0.1 mg./cu.m. threshold limit can be interpreted from the results of repeated feeding to animals and by analogy with hydrogen selenide. This is probably low enough to prevent injury.

Sodium hydroxide. Elkins (1950, p. 84) states inhalation of mists result in upper respiratory tract irritation leading to ulceration. Patty (1949, p. 561) on the basis of experience with caustic mists from 1 to 40 mg./cu.m., concludes a concentration of 2 mg./cu.m. is noticeably but not excessively irritating.

The most important effect of sodium hydroxide mist or dust inhalation is upper respiratory tract irritation, leading to ulceration. The 2 mg./cu.m. threshold limit

can be interpreted from observations upon exposed workmen. It appears low enough to prevent injury.

Stibine. Webster (1946) finds stibine a lung injurant and powerful hemolytic agent, injuring liver and kidney as well. Some animals die after one hour inhalation of 40 ppm. Its action resembles that of the better understood arsine.

The most important effect of stibine inhalation is acute poisoning, largely lung edema. The 0.1 ppm threshold limit can be interpreted by analogy with arsine. It appears low enough to prevent injury.

Stoddard Solvent. Nelson, Ege, Ross, Woodman and Silverman (1943) found 400 ppm produced no marked effects on unacclimated subjects. This solvent is toxicologically identical with gasoline and remarks under that material apply.

Strychnine. McNally (1937) reports that a human death has resulted from swallowing 30 milligrams. It is a convulsive poison.

The most important effect of strychnine inhalation is acute poisoning. The 0.15 mg./cu.m. tentative threshold limit can be interpreted from the known human fatal dose. It corresponds to a maximum intake of 1.5 milligrams in a working day, apparently low enough to prevent injury.

Styrene monomer. Spencer, Irish, Adams and Rowe (1942) found immediate animal death was anesthetic, delayed death was due to lung injury. Blood cells were not affected. Repeated inhalation of 650 ppm had no effect on animals. Humans found 1300 ppm extremely irritating to eye and nose, and 400 ppm had an objectionable odor but little irritation. Carpenter, Shaffer, Weil and Smyth (1944) found irritation and early narcosis in humans at 800 ppm.

The most important effect of styrene vapor inhalation is narcosis. The threshold limit of 200 ppm can be interpreted from results of repeated animal inhalations and human sensory response. It is low enough to prevent definite narcosis.

Sulfur dioxide. Kehoe, Machle, Kitzmiller and LeBlanc (1932) studied many workmen continuously exposed to sulfur dioxide and found only upper respiratory tract chronic irritation. Henderson and Haggard (1943, p. 131) conclude it is an irritant without systemic effect. They give 400 to 500 ppm as dangerous in a short time, 20 ppm irritating the eye and causing coughing, 8 to 12 ppm irritating the throat, and 3 to 5 ppm detectable by odor. Elkins (1950, p. 81) finds slight human irritation at 2 ppm and objectionable irritation at 10 to 30 ppm.

The most important effect of sulfur dioxide inhalation is respiratory tract irritation, with lung edema or respiratory arrest the maximum effect. The 10 ppm threshold limit can be interpreted from human sensory data and examination of exposed workmen. It is low enough to prevent injury.

Sulfur hexafluoride. Lester and Greenberg (1950) found that it is a physiologically inert gas, rats inhaling 800,000 ppm in oxygen for 16 to 24 hours were not affected.

The most important effect of sulfur hexafluoride inhalation is asphyxia from very high concentrations. The 1000 ppm threshold limit can be interpreted from the results of prolonged animal inhalations. It is so far below any possible injurious level that it represents good engineering control, rather than a hazard limit.

Sulfuric acid. Fairhall (1949, p. 83) considers it an upper respiratory tract irritant with lung injury possible. It is corrosive to the skin and eyes, and erodes the teeth. Amdur, Silverman and Drinker (1952) with normal human subjects, found changes in respiration at a concentration as low as 0.35 mg./cu.m., and pronounced decrease in minute volume at 5 mg./cu.m. Elkins (1950, p. 82) considers concentrations above 1 ppm (4 mg./cu.m.) are irritating. Sterner (1943) considered 5 mg./cu.m. as tolerable.

The most important effect of sulfuric acid inhalation is respiratory tract irritation, with lung edema possible. The 1 mg./cu.m. threshold limit can be interpreted from human sensory and physiological data. It is low enough to prevent injury.

Sulfur monochloride. Henderson and Haggard (1943, p. 130) consider it an upper respiratory tract irritant through release of hydrochloric acid, but rarely a lung injurant. Mice die from one minute inhalation of 150 ppm, cats from 15 minutes at 48 ppm. Fairhall (1949, p. 160) concludes chronic systemic effects do not occur. Elkins (1950, p. 81) found 2 to 9 ppm mildly irritating to humans.

The most important effect of sulfur monochloride vapor inhalation is respiratory

tract irritation. The 1 ppm threshold limit can be interpreted from results of single animal inhalations and human sensory data. It is low enough to prevent injury.

Sulfur pentafluoride. Greenberg and Lester (1950) found this to be a lung injurant. Rat lungs were severely injured by one hour at 10 ppm, less severely injured at 1 ppm and not affected at 0.1 ppm. Sixteen hours at 1 ppm was lethal, due to lung injury, while 18 hours at 0.5 ppm injured lungs but did not kill.

The most important effect of sulfur pentafluoride vapor inhalation is lung injury. The 0.025 ppm threshold limit can be interpreted from results of repeated animal inhalation. It is probably low enough to prevent injury.

TEDP. This cholinesterase inhibitor is about half as toxic in single doses to animals as parathion, and the class of cholinesterase inhibiting compounds manifests little tendency to chronic effect.

The most important effect of TEDP inhalation is the reduction of blood cholinesterase. The 0.2 mg./cu.m. threshold limit can be interpreted only by analogy with parathion. It appears low enough to prevent injury.

Tellurium. Steinberg, Massari, Miner and Rink (1942) examined workmen exposed to tellurium fume ranging from 0.01 to 0.1 mg./cu.m., with a peak of 0.74 mg./cu.m. in one sample. The symptoms found were garlic odor of breath and sweat, dryness of mouth, metallic taste and somnolence. No signs of poisoning were found. Excessive absorption would have been shown by gastro-intestinal disturbances, reduction in red blood cells, diminished reflexes, and tremor.

The most important effect of inhalation of tellurium-containing dusts is chronic poisoning centering in the liver. The 0.1 mg./cu.m. threshold limit can be interpreted from the results of examination of exposed workmen. It appears low enough to prevent injury.

TEPP. This cholinesterase inhibitor is about twice as toxic in single doses to animals as parathion, and the class of cholinesterase inhibiting compounds manifests little tendency to chronic effect. Vigliani and Zurlo (1955) on the basis of cholinesterase reduction in workmen, consider 0.0007 mg./cu.m. a satisfactory threshold limit.

The most important effect of TEPP inhalation is the reduction of blood cholinesterase. The 0.05 mg./cu.m. threshold limit can be interpreted only by analogy with parathion. It appears low enough to prevent injury.

p-Tertiary butyl toluene. Hine et al. (1954) in repeated animal inhalations, found narcosis, respiratory tract irritation, liver and kidney changes, blood cell changes like those from benzene, and degenerations in spinal cord and brain. Rats are killed in one hour by about 900 ppm, and slight evidence of effect was found in animals repeatedly inhaling 25 ppm. Humans detect 5 ppm by odor, 80 ppm was unpleasantly irritating and some giddiness was noted at 160 ppm.

The most important effect of p-tertiary butyl toluene inhalation appears to be chronic toxicity, combining effect on blood cells, central nervous system, liver and kidney. The 10 ppm threshold limit can be interpreted from results of repeated animal inhalations and human sensory data. It appears to be low enough to prevent significant toxic effect.

1,1,2,2-Tetrachloroethane. Fairhall (1949, p. 440) concludes this is the most toxic chlorinated hydrocarbon, nine times as toxic as carbon tetrachloride. It is a narcotic and produces liver damage, polyneuritis and white blood cell changes. Elkins (1950, p. 139) refers to an unpublished report of illness from a concentration below 10 ppm. Smyth (1937-55) found rats survive four hours at 500 ppm but are killed by 1000 ppm.

The most important effect of 1,1,2,2-tetrachloroethane inhalation is chronic poisoning centering in the liver. The 5 ppm threshold limit can be interpreted from industrial experience. It is uncertain whether it is low enough to prevent some degree of injury.

Tetrahydrofuran. Lehman and Flury (1943, p. 269) report it a narcotic, irritating mucous membrane and injuring the kidneys. In animals 3400 ppm for eight hours daily for 20 days caused some mucous membrane irritation and light narcosis, with albuminuria, lung and kidney injury and lung irritation in one animal each. ACGIH (1955b) quotes John A. Zapp, Jr. to the effect that repeated inhalation of 200 ppm, then 400 ppm, slightly affected the pulse pressure of dogs, but resulted in no histopathology. Hoffmann and Oettell (1954) in rabbits and cats found some narcosis and mucosal irritation after six hours

inhalation of 3400 ppm. There was no liver or kidney injury from inhalation of even 60,000 ppm.

The most important effect of tetrahydrofuran inhalation is narcosis, with less important injury to liver and kidneys. The 200 ppm tentative threshold limit can be interpreted from results of repeated animal inhalation studies. It appears low enough to prevent injury.

Tetranitromethane. Sievers, Rushing, Gay and Monaco (1947) found in cats irritation of eyes, upper respiratory tract, lung edema, methemoglobinuria and injury to liver and kidney. Concentrations of 3.3 to 25.2 ppm were severely injurious, while 0.1 to 0.4 ppm caused only mild irritation in two exposures. Horn (1954) found in repeated inhalation of 6.35 ppm that some rats died from pneumonia. The effect on dogs was transient anorexia.

The most important effect of tetranitromethane inhalation is poisoning, accompanied by irritation. The 1 ppm threshold limit can be interpreted from results of repeated animal inhalation. It is low enough to prevent injury, but not to prevent all irritation.

Tetryl. Bergman (1952) summarizes ten years' experience in an ordnance plant with more than 1000 exposed workers and concentrations kept below 1.5 mg./cu.m. by constant diligence. Skin sensitization was frequent but no systemic poisoning was found. Hardy and Maloof (1950) report two fatal and eight non-fatal cases with liver injury the most prominent effect. There was upper respiratory tract irritation, very frequent skin sensitization and a few asthmatic sensitizations. Concentrations were as high as 17.7 mg./cu.m.

The most important effect of tetryl inhalation is chronic poisoning like that of trinitrotoluene, but the most frequent effect is sensitization. The 1.5 mg./cu.m. threshold limit can be interpreted from experience with exposed workmen. It is low enough to prevent injury but not sensitizations.

Thallium. Fairhall (1949) reviewing the literature, concludes that thallium has a chronic toxicity greater than that of lead. Useful quantitative data on inhalation appear to be lacking.

The most important effect of thallium inhalation is chronic poisoning. The 0.15 mg./cu.m. tentative threshold limit appears to be based on a quantitative analogy with lead. Its propriety cannot be judged.

Thiram. Meagre data have been found on this substance. Smyth (1937-55) found the rat oral LD_{50} to be 1.30 gm./kg. Rats survived four hours inhalation of a dense dust cloud, unmeasured but estimated to be at least 500 mg./cu.m., with no effect but some brief retardation of growth.

The most important effect of inhalation of thiram appears to be an ill-defined chronic toxicity. The 5 mg./cu.m. tentative threshold limit cannot be interpreted from published data found by the writer, but it appears to be reasonable.

Titanium dioxide. Fairhall (1949, p. 180) concludes titanium dioxide is chemically inert and is not toxic. Lenzi (1936) found a pneumoconiosis in guinea pig lungs after prolonged inhalation of high concentrations.

Inhaled titanium dioxide acts as an inert dust, with only a slight tendency to produce pneumoconiosis. The 15 mg./cu.m. threshold limit is an arbitrary figure uniformly applied to inert nuisance dusts. It appears low enough to prevent injury.

Toluene. Von Oettingen, Neal and Donahue (1942) in repeated exposure of animals, found no blood cell changes or other toxic effects at 800 ppm. Humans inhaling 200 ppm for an eight-hour period found the earliest signs of impaired coordination and lengthened reaction time, while the effects were more prominent and more prompt at 600 to 800 ppm. Fairhall (1949, p. 447) concludes it is a narcotic with irritating properties, but manifests no chronic effects. Elkins (1950, p. 108) cites Greenburg's examination of over 100 workers in atmospheres of 100 to 1100 ppm without marked symptoms. Smyth (1937-55) found rats survive four hours at 4000 ppm, but die from 16,000 ppm. Commercial toluene may contain significant amounts of benzene and may be more toxic than these data indicate.

The most important effect of toluene inhalation is narcosis. The 200 ppm threshold limit can be interpreted from results of repeated animal inhalation, studies of human narcosis and examination of exposed workmen. It appears to be low enough to prevent all effects except the earliest signs of narcosis.

O-Toluidine. Henderson and Haggard (1943, p. 228) quote slight symptoms after several hours at 6 to 23 ppm, and for aniline they give 7 to 53 ppm. Fairhall (1949, p. 450) find symptoms are like those from aniline. Smyth (1937-55) found rats not killed by eight hours inhalation of substantially saturated vapors.

The most important effect of o-toluidine inhalation is poisoning like that from aniline. The 5 ppm threshold limit can be interpreted by analogy with aniline. It appears low enough to prevent injury.

Trichloroethylene. Morse and Goldberg (1943) found headache, nausea and dizziness common in industrial exposures well under the 200 ppm figure proposed by USPHS (1943) on the basis of European opinion. Adams, Spencer, Rowe, McCollister and Irish (1951) found no adverse effects from repeated inhalation by monkeys at 400 ppm, in rats and rabbits at 200 ppm, or in guinea pigs at 100 ppm. Single massive exposures kill by anesthesia. Even 3000 ppm inhaled repeatedly causes no more than very minor organic changes in the liver. They found almost identical effects from single and repeated inhalation of a single concentration, and concluded the effects are chiefly those of narcosis. Voluntary habituation has been found in industry.

The most important effect of trichloroethylene inhalation is narcosis. The 200 ppm threshold limit can be interpreted from the results of repeated animal inhalations and observations in industry. It is not low enough to prevent significant narcosis.

Trichloronaphthalene. After extensive inhalation studies with rats, Drinker (1939) concluded injury is exclusively in the liver and that the permissible limit for repeated inhalation is 5 mg./cu.m. The solid material or its oil solution penetrates the skin and repeated contact leads to chloracne.

The most important effect of trichloronaphthalene inhalation is chronic poisoning centering in the liver. The 5 mg./cu.m. threshold limit can be interpreted from the results of repeated animal inhalations. It is low enough to prevent injury.

Trifluoromonobromomethane. ACGIH (1955b) quotes Chemical Corps Medical Laboratories Research Report No. 180, 1953. This shows that the material is narcotic at high concentrations, but that 23,000 ppm for 18 weeks had no toxic or lung injuring effects on dogs or rats.

The most important effects of trifluoromonobromomethane inhalation are narcosis and respiratory tract irritation. The 1000 ppm threshold limit can be interpreted from the results of repeated animal inhalations. It appears low enough to prevent injury.

Trinitrotoluene. Von Oettingen et al (1944) in experiments on dogs found only tracheal irritation and some effects on red blood cells from daily intratracheal insufflation of 50 mg./kg. This daily dosage by mouth did not kill in three months but resulted in definite central nervous system, liver and blood cell effects. The material penetrates the skin. Eddy (1944) reports fatal aplastic anemia in three humans at concentrations of 1 to 3.5 mg./cu.m.

The most important effect of trinitrotoluene inhalation is chronic poisoning marked by central nervous system, liver and red blood cell changes. The 1.5 mg./cu.m. threshold limit can be interpreted from a report of industrial fatality. It is probably not low enough to prevent all injuries.

Turpentine. Smyth and Smyth (1928) found that repeated inhalation of 750 ppm did not injure animals. Nelson, Ege, Ross, Woodman and Silverman (1943) with unacclimated subjects found nose and throat irritation at 75 ppm, while 175 ppm was judged intolerable. Fairhall (1949, p. 464) notes irritation, narcosis, and kidney injury.

The most important effect of turpentine inhalation is narcosis, but irritation of the respiratory tract is more frequently encountered. The 100 ppm threshold limit can be interpreted from results of repeated animal inhalations and human sensory data. It is low enough to prevent injury.

Uranium (soluble). Uranium is a radiological hazard through emission of alpha particles, and a toxicological hazard primarily through injury to kidney tubules. Hodge, Stokinger and Neuman (1949) show that on inhalation, the toxicological hazard is much greater than the radiological hazard. In repeated animal experiments, they find that 0.5 mg./cu.m. allows a reasonable margin of safety over the level producing kidney injury.

The most important effect of inhalation of soluble uranium dusts is chronic poisoning, centering in the kidney. The 0.05

mg./cu.m. threshold limit can be interpreted from the results of repeated animal inhalations. It is low enough to prevent injury.

Uranium (insoluble). Hodge, Stokinger, Neuman, Bale and Brandt (1949) show that on inhalation, the toxicological action of uranium on the kidney is much more a hazard than the alpha radiation hazard of the uranium stored in the bones. In repeated animal experiments, they find 0.20 to 0.25 mg./cu.m. allows reasonable margin of safety over the level producing kidney injury.

The most important effect of inhalation of insoluble uranium dusts is chronic poisoning centering in the kidney. The 0.25 mg./cu.m. threshold limit can be interpreted from the results of repeated animal inhalations. It is low enough to prevent injury.

Vanadium. Roshchin (1952) exposed rats to vanadium pentoxide fume of 0.3 to 0.5 mg./cu.m. for two hours every other day for three months, and to dust at 1 to 3 mg./cu.m. for one hour daily for four months. They lost some weight and had bloody nasal secretion. Some evidence of pulmonary edema was seen from the fume. Acutely 8 mg./cu.m. of the dust was injurious in one hour and 70 to 80 mg./cu.m. was lethal. The author suggests a threshold limit of 0.1 mg./cu.m. for fume and 0.5 mg./cu.m. for dust.

The most important effect of inhalation of vanadium dusts is bronchial and lung injury. The threshold limits of 0.5 mg./cu.m. for dust and 0.1 mg./cu.m. for fume can be interpreted from the results of repeated animal inhalations. They appear low enough to prevent injury.

Vinyl chloride. Patty, Yant and Waite (1930) found no serious disturbance in guinea pigs inhaling 5000 ppm for several hours. Higher concentrations produced only narcosis. In humans, 50,000 ppm is noticed as a slight odor and nose irritation, and dizziness is evident.

The most important effect of vinyl chloride inhalation is narcosis. The 500 ppm threshold limit can be interpreted from the results of single animal inhalations and human response. It appears low enough to prevent significant narcosis.

Warfarin. Saunders, Heisey, Goldstone and Bay (1955) found injection of 0.5 mg./kg. for five days killed most of a small group of rats, while a single injection of 100 mg./kg. caused only 30% mortality. One human fatality arose from the consumption of a total of 750 mg. over a 15-day period.

The most important effect of warfarin inhalation is chronic poisoning centering in the blood coagulation mechanism. The 0.5 mg./cu.m. tentative threshold limit cannot be interpreted in quantitative terms. It involves a maximum daily absorption of 5 mg., about one-tenth the amount which was fatal to one man.

Xylene. Nelson, Ege, Ross, Woodman and Silverman (1943) found 200 ppm definitely irritating to eye, nose and throat. Fairhall (1949, p. 468) concludes the effects are like those of toluene, narcosis without damage to red blood cells.

The most important effect of xylene inhalation is narcosis. The 200 ppm threshold limit can be interpreted from human sensory data. It is irritating to eye, nose and throat. There are no data to show whether or not significant narcosis occurs at this concentration, but since narcotic activity is greater than that of toluene it is to be anticipated.

Zinc oxide fume. Drinker, Thomson and Finn (1927b) reported that experimental fume fever from zinc oxide in man results from excessive inhalation, but does not occur below 15 mg./cu.m. In industry it was found that 14 mg./cu.m. caused no reaction after eight hours, and in the laboratory 45 mg./cu.m. was without effect in 20 minutes. This condition is a transient fever with chills, muscular pains, nausea and vomitting. An immunity is apparently built up.

The most important effect of zinc oxide fume inhalation is transient metal fume fever. The 15 mg./cu.m. threshold limit can be interpreted from the results of extensive human experiment and experience in industry. It is low enough to prevent injury.

Zirconium. ACGIH (1955b) quotes unpublished data from the University of Rochester A.E.C. Project. Four species of animals inhaled the following, all expressed in terms of contained zirconium: Zirconium oxide, 1.5 micron dust, 75 mg./cu.m. for 30 days, 11 mg./cu.m. for 60 days, 3.5 mg./cu.m. for one year; zirconium tetrachloride, 0.6 micron dust, 6 mg./cu.m. for 60 days, 3.5 mg./cu.m. for a year. Only the higher concentration of tetrachloride had any effect, presumably due to liberated hydrochloric acid.

The effect of zirconium dust inhalation is that of an inert nuisance dust when insoluble, and possibly bronchial and lung irritation when soluble. The 5 mg./cu.m. tentative threshold limit can be interpreted from the results of repeated animal inhalations. It appears low enough to prevent injury.

Bibliography

ACGIH (1947): 1947 M.A.C. values. *Ind. Hyg. Newsletter*, 7:15-16, August.

ACGIH (1948): Threshold limit values adopted at April, 1948 meeting (privately circulated).

ACGIH (1949): Threshold limit values adopted at April 1949 meeting (privately circulated).

ACGIH (1950): Threshold limit values. *Arch. Ind. Hyg. & Occup. Med.*, 2:98-100.

ACGIH (1951): Threshold limit values for 1951. *Arch. Ind. Hyg. & Occup. Med.*, 4:398-400.

ACGIH (1952): Threshold limit values for 1952. *Arch. Ind. Hyg. & Occup. Med.*, 6:178-180.

ACGIH (1953): Threshold limit values for 1953. *Arch. Ind. Hyg. & Occup. Med.*, 8:296-298.

ACGIH (1953b): Privately circulated document giving support for many earlier adopted threshold limits, and those proposed in 1953.

ACGIH (1954): Threshold limit values for 1954. *Arch. Ind. Hyg. & Occup. Med.*, 9:530-534.

ACGIH (1954b): Privately circulated document giving support for threshold limits newly proposed in 1954.

ACGIH (1955): Threshold limit values for 1955. *AMA Arch. Ind. Health;* 11:521-524.

ACGIH (1955b): Privately circulated document giving support for threshold limits newly proposed in 1955.

ACGIH (1956): Proposed threshold limit values for 1956, presented April 23, 1956 to the Philadelphia, annual meeting. Privately circulated in advance of publication.

ADAMS, E. M.; SPENCER, H. C., and IRISH, D. D. (1940): The acute vapor toxicity of allyl chloride. *J. Ind. Hyg. & Tox.*, 22:79-86.

ADAMS, E. M.; SPENCER, H. C.; ROWE, V. K., and IRISH, D. D. (1950): Vapor toxicity of 1,1,1-trichloroethane determined by experiments on laboratory animals. *Arch. Ind. Hyg. & Occup. Med.*, 1:225-236.

ADAMS, E. M.; SPENCER, H. C.; ROWE, V. K.; MCCOLLISTER, D. D. and IRISH, D. D. (1951): Vapor toxicity of trichloroethylene determined by experiments on laboratory animals. *Arch. Ind. Hyg. & Occup. Med.*, 4:469-481.

ADAMS, E. M.; SPENCER, H. C.; ROWE, V. K.; MCCOLLISTER, D. D., and IRISH, D. D. (1952): Vapor toxicity of carbon tetrachloride determined by experiments on laboratory animals. *Arch. Ind. Hyg. & Occup. Med.*, 6:50-66.

AERO MEDICAL ASSOCIATION (1953), Committee on Aviation Toxicology: *Aviation Toxicology*, Blakiston, New York.

AHLMARK, A. (1948): Poisoning by methyl mercury compounds. *Brit. J. Ind. Med.*, 5:117-119.

ALVAREZ, W. C., and HYMAN, S. (1953): Absence of toxic manifestations in workers exposed to chlordane. *Arch. Ind. Hyg. & Occup. Med.*, 8:480-483.

AMA (1951) Council on Pharmacy and Chemistry: Toxic effects of technical benzene hexachloride and its principal isomers. *J.A.M.A.*, 147:571-574.

AMDUR, M. O.; SILVERMAN, L., and DRINKER, P. (1952): Inhalation of sulfuric acid mist by human subjects. *Arch. Ind. Hyg. & Occup. Med.*, 6:305-313.

ASHE, W. F.; LARGENT, E. J.; DUTRA, F. R.; HUBBARD, D. M., and BLACKSTONE, M. (1953): Behavior of mercury in the animal organism following inhalation. *Arch. Ind. Hyg. & Occup. Med.*, 7:19-43.

Baltimore City Health Department (1943), Division of Industrial Hygiene: *Baltimore Health News*, 20: September, 1943.

BARNES, J. M. (1953): Toxic hazards of certain pesticides to man. World Health Organization, Geneva. Monograph 16.

BARTHELEMY, H. L. (1939): Ten years experience with industrial hygiene in connection with the manufacture of viscose rayon. *J. Ind. Hyg. & Tox.*, 21:141-151.

BERGMAN, B. B. (1952): Tetryl toxicity. *Arch. Ind. Hyg. & Occup. Med.*, 5:10-20.

BLOOMFIELD, J. J., and BLUM, W. (1928): Health hazards in chromium plating. U.S. Public Health Service, *Pub. Health Rpts.*, 43:2330-2351.

BRADLEY, W. R., and FREDRICK, W. G. (1941): The toxicity of antimony: Animal studies. *Ind. Med.*, 10, Ind. Hyg. Sec., 2:15-22.

BRANDT, A. D. (1947): Industrial Health Engineering. Wiley, New York.

BRIEGER, H., and HODES, W. A. (1951): Toxic effects of exposure to vapors of aliphatic amines. *Arch. Ind. Hyg. & Occup. Med.*, 3:287-291.

BRIEGER, H.; SEMISCH, C. W.; STASNEY, J., and PIATNEK, D. A. (1954): Industrial antimony poisoning. *Ind. Med.*, 23:521-523.

BROWN, H. V., and BUSH, A. F. (1950): Parathion inhibition of cholinesterase. *Arch. Ind. Hyg. & Occup. Med.*, 1:633-636.

BUCHAN, R. F. (1947): Industrial selenosis. *Occup. Med.*, 3:439-436.

CAMERON, G. R.; THOMAS, J. C.; ASHMORE, S. A.; WARREN, E. H.; BUCHAN, J. L., and MCKENNY-HUGHES, A. W. (1937): The toxicity of certain chlorine derivatives of benzene, with special reference to o-dichlorobenzene. *J. Path. Bact.*, 44:281-296.

CARPENTER, C. P. (1937). The chronic toxicity of tetrachloroethylene. *J. Ind. Hyg. & Tox.*, 19:323-336.

CARPENTER, C. P.; SHAFFER, C. B.; WEIL, C. S., and SMYTH, H. F., JR. (1944): Studies on the inhalation of 1:3-Butadiene. *J. Ind. Hyg. & Tox.*, 26:69-78.

CARPENTER, C. P.; SMYTH, H. F., JR., and SHAFFER, C. B. (1948): The acute toxicity of ethylene imine to small animals. *J. Ind. Hyg. & Tox.*, 30:2-6.

CARPENTER, C. P.; WEIL, C. S.; POZZANI, U. C., and SMYTH, H. F., JR. (1950): Comparative acute and subacute toxicities of allethrin and pyrethrins. *Arch. Ind. Hyg. & Occup. Med.*, 2:420-432.

CARPENTER, C. P.; POZZANI, U. C., and WEIL, C. S. (1953): Toxicity and hazard of diisobutyl ketone vapors. *Arch. Ind. Hyg. & Occup. Med.*, 8:377-381.

Committee on Chemical Agents (1949). Report of the panel on Environmental Agents, Ninth Annual Congress on Industrial Health, Chicago, January 18-19, 1949. Published in *Arch. Ind. Hyg. & Occup. Med.*, 1:601-624, 1950.

COMSTOCK, C. C.; MACNAMEE, J. K.; OZBURN, E. E.; FOGLEMAN, R. W., and OBERST, F. W. (1952): Monochloromonobromomethane; inhalation toxicity, pathology and symptomatology in rats and mice. Chemical Corps Medical Laboratories, Research Report No. 113.

COMSTOCK, C. C., and OBERST, F. W. (1953): The median detectable concentration of diborane, pentaborane and decaborane by odor for man. Chemical Corps Medical Laboratories, Research Report No. 206.

COMSTOCK, C. C.; LAWSON, L. H.; GREENE, E. A., and OBERST, F. W. (1954): Inhalation toxicity of hydrazine vapor. *Arch. Ind. Hyg. & Occup. Med.*, 10:476-490.

COOK, W. A. (1945): Maximum allowable concentrations of industrial atmospheric contaminants. *Ind. Med.*, 14:936-946.

CULVER, D.; CAPLAN, P., and BATCHELOR, G. S. (1956): Studies of human exposure during aerosol application of malathion and chlorthion. *AMA Arch. Ind. Health*, 13:37-50.

DEICHMANN, W. B.; KITZMILLER, K. V., and WITHERUP, S. (1944): Phenol studies VII. Chronic phenol poisoning, with special reference to the effects upon experimental animals of the inhalation of phenol vapor. *Am. J. Clin. Path.*, 14:273-277.

DERNEHL, C. U.; NAU, C. A., and SWEETS, H. H. (1945): Animal studies on the toxicity of inhaled antimony trioxide, *J. Ind. Hyg. & Tox.*, 27:256-262.

DERNEHL, C. U. (1951): Clinical experiences with exposures to ethylene amines. *Ind. Med. & Surg.*, 20:541-546.

DIEKE, S. H., and RICHTER, C. P. (1946): Comparative assays of rodenticides on wild Norway rats. U.S. Public Health Service, *Pub. Health Rpts.*, 61:672-679.

DIERKER, H., and BROWN, P. G. (1944): Study of a fatal case of ethylene chlorhydrin poisoning. *J. Ind. Hyg. & Tox.*, 26:277-279.

DONLEY, D. E. (1936): Toxic encephalopathy and volatile solvents in industry. *J. Ind. Hyg. & Tox.*, 18:571-577.

DRINKER, C. K. (1939): Further observations on the possible systemic toxicity of certain of the chlorinated hydrocarbons. *J. Ind. Hyg. & Tox.*, 21:155-159.

DRINKER, P.; THOMSON, R. M., and FINN, J. L. (1927): Metal fume fever. III. The effects of inhaling magnesium oxide fume. *J. Ind. Hyg.*, 9:187-192.

DRINKER, P.; THOMSON, R. M., and FINN, J. L. (1927b): Metal fume fever. IV. Threshold doses of zinc oxide, preventive measures and the chronic effects of repeated exposures. *J. Ind. Hyg.*, 9:331-345.

DRINKER, P.; YAGLOU, C. P., and WARREN, M. F. (1943): The threshold toxicity of gasoline vapor. *J. Ind. Hyg. & Tox.*, 25:225-232.

DUDLEY, H. C., and MILLER, J. W. (1941): Effects of subacute exposure to hydrogen selenide. *J. Ind. Hyg. & Tox.*, 23:470-477.

DUDLEY, H. C., and NEAL, P. A. (1942): Toxicology of acrylonitrile. I. A study of the acute toxicity. *J. Ind. Hyg. & Tox.*, 24:27-36.

DUDLEY, H. C.; SWEENEY, T. R., and MILLER, J. W. (1942). Toxicology of acrylonitrile. II. Studies of effects of daily inhalation. *J. Ind. Hyg. & Tox.*, 24:255-258.

EDDY, J. H., JR. (1944): Aplastic anemia following trinitrotoluene exposure. *J.A.M.A.*, 125:1169-1172.

ELKINS, H. B. (1950): The chemistry of industrial toxicology. Wiley, New York.

FAIRHALL, L. T., and MILLER, J. W. (1941): A study of the relative toxicity of the molecular components of lead arsenate. U. S. Public Health Service, *Pub. Health Rpts.*, 56:1610-1625.

FAIRHALL, L. T.; MILLER, J. W., and WEAVER, F. L. (1943): The effect of arsenates on the storage of lead. U.S. Public Health Service, *Pub. Health Rpts.*, 58:955-959.

FAIRHALL, L. T.; DUNN, R. C.; SHARPLESS, N. E., and PRITCHARD, E. A. (1945): The toxicity of molybdenum. U.S. Public Health Service, *Pub. Health Bull.* 293.

FAIRHALL, L. T. (1949): Industrial Toxicology. Williams & Wilkins, Baltimore.

FAIRLEY, A.; LINTON, E. C., and FORD-MOORE, A. H. (1934): The toxicity to animals of 1,4-dioxane. *J. Hyg.*, 34:486-501.

FEINER, B.; BURKE, W. J., and BALIFF, J. (1946): An industrial hygiene survey of an onion dehydrating plant. *J. Ind. Hyg. & Tox.*, 28:278-279.

FIELDNER, A. C.; KATZ, S. H., and KINNEY, S. P. (1921): Gas masks for gases met in fighting fires. U.S. Bur. Mines Tech. Paper 248.

FIELDNER, A. C.; SAYERS, R. R.; YANT, W. P.; KATZ, S. H.; SHOHAN, J. B., and LEITCH, R. D. (1931): Warning agents for fuel gases. U.S. Bur. Mines, Monograph #4.

FINE, E. A., and WILLS, J. H. (1950): Pharmacologic studies on furfuryl alcohol. *Arch. Ind. Hyg. & Occup. Med.*, 1:625-632.

FITZHUGH, O. G., and NELSON, A. A. (1947): Chronic oral toxicity of alpha-naphthyl-thiourea. *Proc. Soc. Exper. Biol. Med.*, 64:305-310.

FLINN, R. H.; NEAL, P. A., and FULTON, W. B. (1941): Industrial manganese poisoning. *J. Ind. Hyg. & Tox.*, 23:374-387.

FLURY, F., and ZERNIK, F. (1931): Schädliche Gase und Dämpfe. J. Springer, Berlin.

GOLDBLATT, M. W. (1944): Toxic effects of ethylene chlorhydrin. II—Experimental. *Brit. J. Ind. Med.*, 1:213-223.

GOLDBLATT, M. W., and CHIESMAN, W. E. (1944): Toxic effects of ethylene chlorhydrin. I—Clinical. *Brit. J. Ind. Med.*, 1:207-213.

GRAY, E. LEB., MACNAMEE, J. K., and GOLDBERG, S. B. (1952): Toxicity of NO_2 vapors at very low levels. *Arch. Ind. Hyg. & Occup. Med.*, 6:20-21.

GREENBERG, L. A., and LESTER, D. (1950): The toxicity of sulfurpentafluoride. *Arch. Ind. Hyg. & Occup. Med.* 2:350-352.

GREENBERG, L.; MAYERS, M. R.; GOLDWATER, L. J.; BURKE, W. J., and MOSKOWITZ, S. (1938): Health hazards in the manufacture of fused collars. I—Exposure to ethylene glycol monomethyl ether. *J. Ind. Hyg. & Tox.*, 20:134-147.

HAAG, H. B.; FINNEGAN, J. K.; LARSON, P. S.; RIESE, W., and DREYFUSS, M. L. (1950): Comparative chronic toxicities for warm-blooded animals. of 2,2-bis-(p-chlorophenyl)-1,1,1-trichloroethane (DDT) and 2,2-bis-(p-methoxyphenyl)-1,1,1-trichloroethane (DMDT, methoxychlor). *Arch. Intern. Pharmacodynamie*, 83:491-504.

HAGGARD, H. W.; GREENBERG, L. A., and TURNER, J. M. (1944): The physiological principles governing the action of acetone, together with determination of toxicity. *J. Ind. Hyg. & Tox.*, 26:133-151.

HAGGARD, H. W.; MILLER, D. P., and GREENBERT, L. A. (1945): The amyl alcohols and their ketones: their metabolic fates and comparative toxicities. *J. Ind. Hyg. & Tox.*, 27:1-14.

HANZLIK, P. J. (1923): Toxicity and actions of the butyl amines. *J. Pharm. Exptl. Therap.*, 20:435-449.

HARDY, H. L., and MALOOF, C. C. (1950): Evidence of systemic effect of tetryl. *Arch. Ind. Hyg. & Occup. Med.*, 1:545-555.

HAYHURST, E. R. (1936): Poisoning by petroleum distillates. *Ind. Med.*, 5:53-63.

HEMEON, W. C. L. (1955). Plant and process ventilation. The Industrial Press, New York.

HENDERSON, Y.; HAGGARD, H. W.; TEAGUE, M. C.; PRINCE, A. L., and WUNDERLICH, R. M. (1921): Physiological effects of automobile exhaust gas and standards of ventilation for brief exposure. *J. Ind. Hyg.*, 3:79-137.

HENDERSON, Y., and HAGGARD, H. W. (1943): Noxious Gases, 2nd ed. Reinhold, New York.

HEPPEL, L. A.; NEAL, P. A.; PERRIN, T. L.; ORR, M. L., and PORTERFIELD, V. T. (1944): Toxicology of dichloromethane. *J. Ind. Hyg. & Tox.*, 26:8-21.

HEPPEL, L. A.; NEAL, P. A.; HIGHMAN, B., and PORTERFIELD, V. T. (1946): Toxicology of 1,2-dichloropropane. *J. Ind. Hyg. & Tox.*, 28:1-8.

HINE, C. H., *et al.* (1954): Toxicological studies on p-tertiary butyl toluene. *Arch. Ind. Hyg. & Occup. Med.*, 9:227-244.

HODGE, H. C.; STOKINGER, H. E., and NEUMAN, W. F. (1949): Suggested maximum concentration of soluble uranium compounds in air. AECD 2785, n.d., decl. Dec. 1949.

HODGE, H. C.; STOKINGER, H. E.; NEUMAN, W. F.; BALE, W. F., and BRANDT, A. E. (1949): Suggested maximum allowable concentration of insoluble uranium compounds in air. AECD 2784, n.d., decl. 1949.

HODGE, H. C.; MAYNARD, E. A., and BLANCHET, H. J., JR. (1952): Chronic oral toxicity tests of Methoxychlor in rats and dogs. *J. Pharm. Exptl. Therap.*, 104:60-66.

HODGE, H. C.; MAYNARD, E. A.; DOWNS, W.; BLANCHET, H. J., JR., and JONES, C. K. (1952): Acute and Short-term oral toxicity tests of ferric dimethyldithiocarbamate (Ferbam) and zinc dimethyldithiocarbamate (Ziram). *J. Amer. Pharm. Assn., Sci. ed*, 41:662-665.

HODGE, H. C.; MAYNARD, E. A.; HURWITZ, L., DISTEFANO, V.; DOWNS, W. L.; JONES, C. K., and BLANCHET, H. J., JR. (1954): Studies of the toxicity and of the enzyme kinetics of ethyl p-nitrophenyl thionobenzene phosphonate. *J. Pharm. Exptl. Therap.*, 112:29-39.

HOFMANN, and OETTEL, H. (1954): Toxicity of tetrahydrofuran. *Naunyn-Schmiedebergs Arch. Exptl. Pathol. Pharmakol;* 222:233-235. (From *Chem. Abstracts,* 48:7788, 1954).

HOLLINGSWORTH, R. L.; ROWE, V. K.; OYEN, F.; MCCOLLISTER, D. D., and SPENCER, H. C. (1956): Toxicity of ethylene oxide determined on experimental animals. *Arch. Ind. Health,* 13:217-227.

HORN, H. J. (1954): Inhalation toxicology of tetranitromethane. *Arch. Ind. Hyg. & Occup. Med.,* 10:213-222.

HORN, H. J., and WEIR, R. J. (1955): Inhalation toxicology of chlorine trifluoride. *Arch. Ind. Health,* 12:515-521.

HORN, H. J., and WEIR, R. J. (1956): Inhalation toxicology of chlorine trifluoride. *Arch. Ind. Health,* 13:340-345.

HUEPER, W. C. (1950): Carcinogens and carcinogenesis. *Am. J. Med.,* 8:355-371.

I.L.O. (1930): Occupation and health, 2nd vol. International Labour Office, Geneva.

INGLE, L. (1953): The toxicity of chlordane vapors. *Science,* 118:213-214.

INGRAM, F. R. (1951): Methyl bromide fumigation and control in the date-packing industry. *Arch. Ind. Hyg. Occup. Med.,* 4:193-198.

IRISH, D. D.; ADAMS, E. M.; SPENCER, H. C., and ROWE, V. K. (1940): The response attending exposure of laboratory animals to vapors of methyl bromide. *J. Ind. Hyg. & Tox.,* 22:218-230.

JACOBSON, K. H.; HACKLEY, E. B., and FEINSILVER, L. (1956): The toxicity of inhaled ethylene oxide and propylene oxide vapors. *Arch. Ind. Health,* 13:237-244.

JOHNSON, G. A.; FLETCHER, J. H.; NOLAN, K. G., and CASSADAY, J. T. (1952): Decreased toxicity and cholinesterase inhibition in a new series of dithiophosphates. *J. Econ. Entomol.,* 45:279-283.

KEHOE, R. A.; MACHLE, W. F.; KITZMILLER, K., and LEBLANC, T. J. (1932). On the effects of prolonged exposure to sulfur dioxide. *J. Ind. Hyg.,* 14:159-173.

KEHOE, R. A.; DEICHMANN-GRUEBLER, W., and KITZMILLER, K. V. (1939): Toxic effects upon rabbits of pentachlorophenol and sodium pentachlorophenate. *J. Ind. Hyg. & Tox.,* 21:160-172.

KINCAID, J. F.; STRONG, J. S., and SUNDERMAN, F. W. (1953): Nickel poisoning. I—Experimental study of the effects of acute and subacute exposure to nickel carbonyl. *Arch. Ind. Hyg. & Occup. Med.,* 8:48-60.

KRACKOW, E. H. (1953): Toxicity and health hazards of boron hydrides. *Arch. Ind. Hyg. & Occup. Med,* 8:335-339.

LACKEY, R. W. (1949): Observations on the acute and chronic toxicity of toxaphene in the dog. *J. Ind. Hyg. & Tox.,* 31:117-120.

LARGENT, E. J. (1952): Rates of elimination of fluoride stored in the tissues of man. *Arch. Ind. Hyg. & Occup. Med.,* 6:37-42.

LEHMAN, A. J. (1949): Pharmacological considerations of insecticides. *Assoc. Food Drug Off. of U.S.,Q. Bull.* 13:65-70.

LEHMANN, K. B. (1886): Experimentelle Studien über den Einfluss technisch und hygienisch wichtiger Gase und Dämpfe auf den Organismus. *Arch. f. Hyg.,* 5:68.

LEHMANN, K. B., and FLURY, F. (1943): Toxicology and hygiene of industrial solvents. Translated by E. King and H. F. Smyth, Jr. Williams and Wilkins, Baltimore.

LENZI, L. (1936): Pneumoconiosis caused by titanium. *Rass. Med. Applicata Lavoro Ind.;* 7:301-318.

LESTER, D., and GREENBERG, L. A. (1950): The toxicity of sulfur hexafluoride. *Arch. Ind. Hyg. & Occup. Med.,* 2:348-349.

LUNDGREN, K-D., and SWENSSON, A. (1949): Occupational poisoning by alkyl mercury compounds. *J. Ind. Hyg. & Tox.,* 31:190-200.

MACHLE, W.; SCOTT, E. W., and TREON, J. (1939): The phyisological response to isopropyl ether and to a mixture of isopropyl ether and gasoline. *J. Ind. Hyg. & Tox.,* 21:72-96.

MACHLE, W., and EVANS, E. E. (1940): Exposure to fluorine in industry. *J. Ind. Hyg. & Tox.* 22:213-217.

MACHLE, W.; SCOTT, E. W., and TREON, J. (1940): The physiological response of animals to some simple mononitroparaffins and to certain derivatives of these compounds. *J. Ind. Hyg. & Tox.,* 22:315-332.

MACHLE, W.; KITZMILLER, K. V.; SCOTT, E. W., and TREON, J. F. (1942): The effect of inhalation of hydrogen chloride. *J. Ind. Hyg. & Tox.,* 24:222-225.

MACHLE, W.; SCOTT, E. W.; TREON, J. F.; HEYROTH, F. F., and KITZMILLER, K. V. (1945): The physiological response of animals to certain chlorinated mononitroparaffins. *J. Ind. Hyg. & Tox.,,* 27:95-102.

MCCLOSKY, W. T.; SMITH, M. I., and LILLIE, R. D. (1945): Studies on the pharmacologic action and the pathology of alphanaphthylthiourea (ANTU) U.S. Public Health Service, *Pub. Health Rpts.,* 60:1101-1113.

MCCORD, C. P. (1932): The toxicity of allyl alcohol. *J.A.M.A.,* 98:2269-2270.

MCGEE, L. C. (1955): Chlorinated insecticides; toxicity for man. *Ind. Med. and Surg.,* 24:101-109.

MCLAUGHLIN, R. S. (1946): Chemical burns of the human cornea. *Am. J. Ophthal.,* 29:1355-1362.

MCNALLY, W. D. (1937): Toxicology. Industrial Medicine Pub. Co. Chicago, Illinois.

MORSE, K. M., and GOLDBERG, L. (1943): Chlorinated solvent exposures. *Ind. Med.,* 12:706-713.

NAU, C. A. (1948): The accidental generation of arsine gas in an industry. *Southern Med. J.,* 41:341-344.

NEAL, P. A.; FLINN, R. H.; EDWARDS, T. I.; REINHART, W. H.; HOUGH, J. W.; DALLAVALLE, J. M.; GOLDMAN, F. H.; ARMSTRONG, D. W.; GRAY, A. S.; COLEMAN, A. L., and POSTMAN, B. F. (1941): Mercurialism and its control in the felt hat industry. U.S. Public Health Service, *Pub. Health Bull.* 263.

NELSON, K. W.; EGE, J. F., JR.; ROSS, M.; WOODMAN, L. E., and SILVERMAN, L. (1943). Sensory response to certain industrial solvent vapors. *J. Ind. Hyg. Tox.,* 25:282-285.

NUCKOLLS, A. H. (1933): The comparative life, fire and explosion hazards of common refrigerants. Underwriters' Lab. Report, Miscellaneous Hazards 2375.

OBERST, F. W.; COMSTOCK, C. C., and MCGRATH, F. P., (1951): Comparative toxicity of halogenated hydrocarbons proposed for fire extinguishants. *Federation Proc.,* 10:328.

OBERST, F. W.; COMSTOCK, C. C., and HACKLEY, E. B. (1954): Inhalation toxicity of 90% hydrogen peroxide vapor. *Arch. Ind. Hyg. & Occup. Med.,* 10:319-327.

OBERST, F. W.; HACKLEY, E. B., and COMSTOCK, C. C. (1956): Chronic toxicity of aniline vapor (5 ppm) by inhalation. *Arch. Ind. Health,* 13: 379-384.

OGLESBY, F. L. (1956): Personal communication to H. F. Smyth Jr., May 18.

PARSONS,, C. E., and PARSONS, M. E. M. (1938): Toxic encephalopathy and granulopenic anemia due to volatile solvents in industry. *J. Ind. Hyg. & Tox.,* 20:124-133.

PATTY, F. A., and YANT, W. P. (1929): Odor intensity and symptoms produced by commercial propane, butane, pentane, hexane and heptane vapor. U.S. Bur. Mines, Rpts. Investigations 2979.

PATTY, F. A.; YANT, W. P., and WAITE, C. P. (1930): Acute response of guinea pigs to vapors of some new commercial organic compounds. V. Vinyl chloride. U.S. Public Health Service, *Pub. Health Rpts.,* 45:1963-1971.

PATTY, F. A.; SCHRENK, H. H., and YANT, W. P. (1935): Acute response of guinea pigs to vapors of some new

commercial organic compounds. VIII—Butanone. U.S. Public Health Service, *Pub. Health Rpts.*, 50:1217-1228.

PATTY, F. A.; YANT, W. P., and SCHRENK, H. H. (1936): Acute response of guinea pigs to vapors of some new commercial organic compounds. XI—Secondary amyl acetate. U.S. Public Health Service, *Pub. Health Rpts.*, 51:811-819.

PATTY, F. A. (1948-9): Industrial hygiene and toxicology, 2 vols. Interscience, New York.

POLLOCK, L. J., FINKELMAN, I., and ARIEFF, A. J. (1943): Toxicity of pyridine in man. *Arch. Internal Med.*, 71:95-106.

POZZANI, U. C.; WEIL, C. S., and CARPENTER, C. P. (1949), Subacute vapor toxicity and range-finding data for ethyl acrylate. *J. Ind. Hyg. & Tox.*, 31:311-316.

POZZANI, U. C., and CARPENTER, C. P. (1951): Response of rodents to repeated inhalation of vapors of tetraethyl orthosilicate. *Arch. Ind. Hyg. & Occup. Med.*, 4:465-468.

POZZANI, U. C., and CARPENTER, C. P. (1954): Response of rats to repeated inhalation of ethylene diamine vapors. *Arch. Ind. Hyg. & Occup. Med.*, 9:223-226.

PRINCI, F., and SPURBECK, G. H. (1951): A study of workers exposed to the insecticides chlordan, aldrin, dieldrin. *Arch. Ind. Hyg. & Occup. Med.*, 3:64-72.

PRODAN, L. (1932): Cadmium poisoning. *J. Ind. Hyg.*, 14:174-196.

RILEY, E. C., and GOLDMAN, F. H. (1937): Control of chromic acid mists from plating tanks. U.S. Public Health Service. *Pub. Health Rpts.*, 52:172-174.

ROHOLM, K. (1937): Fluorine intoxication. Lewis & Co., London.

ROSHCHIN, I. V. (1952): Hygienic characteristics of industrial vanadium aerosol. *Gig. i. Sanit.*, 11:49-53.

ROWE, V. K.; SPENCER, H. C., and BASS, S. L. (1948): Toxicological studies on certain commercial silicones and hydrolyzable silane intermediates. *J. Ind. Hyg. & Tox.*, 30:332-352.

ROWE, V. K.; SPENCER, H. C.; MCCOLLISTER, D. D.; HOLLINGSWORTH, R. L., and ADAMS, E. M. (1952): Toxicity of ethylene dibromide determined on experimental animals. *Arch. Ind. Hyg. & Occup. Med.*, 6:158-173.

ROWE, V. K.; MCCOLLISTER, D. D.; SPENCER, H. C.; ADAMS, E. D., and IRISH, D. D. (1952): Vapor toxicity of tetrachloroethylene for laboratory animals and human subjects. *Arch. Ind. Hyg. & Occup. Med.*, 5:566-579.

ROWE, V. K., and HYMAS, T. A. (1954): Summary of toxicological information on 2,4-D and 2,4,5-T type herbicides and an evaluation of the hazards to livestock associated with their use. *Am. J. Vetern. Res.*, 15:622-629.

ROWE, V. K.; HOLLINGSWORTH, R. L.; OYEN, F.; MCCOLLISTER, D. D., and SPENCER, H. C. (1956): Toxicity of propylene oxide determined on experimental animals. *Arch. Ind. Health*, 13:228-236.

ROZENDAAL, H. M. (1951): Clinical observations on the toxicology of boron hydrides. *Arch. Ind. Hyg. & Occup. Med.*, 4:257-260.

RUSSELL, A. E.; JONES, R. R.; BLOOMFIELD, J. J.; BRITTEN, R. H., and THOMPSON, L. R. (1933): Lead poisoning in a storage-battery plant. U. S. Public Health Service, *Pub. Health Bull. 205.*

SAUNDERS, J. P.; HEISEY, S. R.; GOLDSTONE, A. D., and BAY, E. C. (1955): Comparative toxicities of Warfarin and some 2-acyl-1,3-indandiones in rats. *Agr. Food Chem.*, 3:762-765.

SAYERS, R. R.; FIELDNER, A. C.; YANT, W. P., and THOMAS, B. G. H. (1927): Experimental studies on the effect of ethyl gasoline and its combustion products. U.S. Bur. Mines, Monograph No. 2.

SAYERS, R. R.; YANT, W. P.; LEVY, E., and FULTON, W. B. (1929): Effects of repeated daily exposure of several hours to small amounts of automobile exhaust gas. U.S. Public Health Service, *Pub. Health Bull. 186.*

SAYERS, R. R.; YANT, W. P.; THOMAS, B. G. H., and BERGER, L. B. (1929): Physiological response attending exposure to vapors of methyl bromide, methyl chloride, ethyl bromide and ethyl chloride. U.S. Public Health Service, *Pub. Health Bull. 185.*

SAYERS, R. R.; FIELDNER, A. C.; YANT, W. P.; LEITCH, R. D., and PEARCE, S. J. (1930): Use of ethyl mercaptan to detect leaks in natural-gas distribution systems. U.S. Bur. Mines Rpts., Investigations 3007.

SAYERS, R. R.; YANT, W. P.; CHORNYAK, J., and SHOAF, H. W. (1930): Toxicity of dichlorodifluoromethane, a new refrigerant. U.S. Bur. Mines Rpts., Investigations 3013.

SAYERS, R. R.; SCHRENK, H. H., and PATTY, F. A. (1936): Acute response of guinea pigs to vapors of some new commercial organic compounds. XII—Normal butyl acetate. U.S. Public Health Service, *Pub. Health Rpts.*, 51:1229-1236.

SAYERS, R. R.; YANT, W. P.; SCHRENK, H. H.; CHORNYAK, J.; PEARCE, S. J.; PATTY, F. A., and LINN, J. G. (1942): Methanol poisoning. I—Exposure of dogs to 450-500 ppm methanol vapor in air. U.S. Bur. Mines Rpts., Investigations 3617.

SCHRENK, H. H.; PATTY, F. A., and YANT, W. P. (1933): Acute response of guinea pigs to vapors of some new commercial organic compounds. VII—Dichloroethyl ether. U.S. Public Health Service, *Pub. Health Rpts.*, 48:1389-1398.

SCHRENK, H. H.; YANT, W. P.; CHORNYAK, J., and PATTY, F. A. (1936): Acute response of guinea pigs to vapors of some new commercial organic compounds. XIII—Methyl formate. U.S. Public Health Service, *Pub. Health Rpts.*, 51:1329-1337.

SCHRENK, H. H.; YANT, W. P., and PATTY, F. A. (1936): Acute response of guinea pigs to vapors of some new commercial organic compounds. X—Hexanone (methyl butyl ketone). U.S. Public Health Service, *Pub. Health Rpts.*, 51:624-631.

SEXTON, R. J., and HENSON, E. V. (1950): Experimental ethylene oxide human skin injuries. *Arch. Ind. Hyg. & Occup. Med.*, 2:549-564.

SIEVERS, R. F., EDWARDS, T. I.; MURRAY, A. L., and SCHRENK, H. H. (1942): Effects of exposure to known concentrations of CO. *J.A.M.A.*, 118:585-588.

SIEVERS, R. F.; RUSHING, E.; GAY, H., and MONACO, A. R. (1947): Toxic effects of tetranitromethane, a contaminant in crude TNT. U.S. Public Health Service, *Pub. Health Rpts.*, 62:1048-1061.

SILVER, S. D., and MCGRATH, F. P. (1948): A comparison of acute toxicities of ethylene imine and ammonia to mice. *J. Ind. Hyg. & Tox.*, 30:7-9.

SILVERMAN, L., SCHULTE, H. F.; and FIRST, M. W. (1946): Further studies on sensory response to certain industrial solvent vapors. *J. Ind. Hyg. & Tox.*, 28:262-266.

SILVERMAN, L.; WHITTENBERGER, J. L., and MULLER, J. (1949): Physiological response of man to ammonia in low concentrations. *J. Ind. Hyg. & Tox.*, 31:74-78.

SJOBERG, S-G. (1951): Health hazards in the production and handling of vanadium pentoxide. *Arch. Ind. Hyg. & Occup. Med.*, 3:631-646.

SKINNER, J. B. (1947): Toxicity of 2-Nitropropane. *Ind. Med.*, 16:441-443.

SKLYANSKAYA, R. M., and RAPPAPORT, J. L. (1935): Chronic chlorine poisoning of rabbits with small doses of chlorine, and the development of the fetus in chlorine-poisoned rabbits. *Arch. exptl. Path. Pharmakol.*, 177:276-287.

SMITH, W. W., and VON OETTINGEN, W. F. (1947): The acute and chronic toxicity of methyl chloride. *J. Ind. Hyg. & Tox.*, 29:47-52.

SMYTH, H. F., and SMYTH, H. F., JR. (1928): Inhalation experiments with certain lacquer solvents. *J. Ind. Hyg.*, 10:261-271.

SMYTH, H. F., JR. (1937-55): Unpublished work by Chemical Hygiene Fellowship, Mellon Institute, Pittsburgh.

SMYTH, H. F., JR., and SEATON, J. (1940): Acute

response of guinea pigs and rats to inhalation of the vapors of tetraethyl orthosilicate. *J. Ind. Hyg. & Tox.*, 22:288-296.

SMYTH, H. F., JR.; SEATON, J., and FISCHER, L. (1942): Response of guinea pigs and rats to repeated inhalation of vapors of mesityl oxide and isophorone. *J. Ind. Hyg. & Tox.*, 24:46-50.

SMYTH, H. F., JR., and CARPENTER, C. P. (1945). Note upon the toxicity of ethylene chlorhydrin by skin absorption. *J. Ind. Hyg. & Tox.*, 27:93.

SMYTH, H. F., JR.; CARPENTER, C. P., and WEIL, C. S. (1951): Range-finding toxicity data, List IV. *Arch. Ind. Hyg. & Occup. Med.*, 4:119-122.

SPECHT, H. (1938): Acute response of guinea pigs to inhalation of methyl isobutyl ketone. U.S. Public Health Service, *Pub. Health Rpts.*, 53:292-300.

SPECHT, H.; MILLER, J. W.; VALAER, P. J., and SAYERS, R. R. (1940): Acute response of guinea pigs to the inhalation of ketone vapors. U.S. Public Health Service, *Natl. Inst. Health Bull.* 176.

SPENCER, H. C.; IRISH, D. D.; ADAMS, E. M., and ROWE, V. K. (1942): The response of laboratory animals to monomeric styrene. *J. Ind. Hyg. & Tox.*, 24:295-301.

SPENCER, H. C.; ROWE, V. K.; ADAMS, E. M., and IRISH, D. D. (1948): Toxicological studies on laboratory animals of certain alkyldinitrophenols used in agriculture. *J. Ind. Hyg. & Tox.*, 30:10-25.

SPENCER, H. C.; ROWE, V. K.; ADAMS, E. M.; MCCOLLISTER, D. D., and IRISH, D. D. (1951): Vapor toxicity of ethylene dichloride determined by experiments on laboratory animals. *Arch. Ind. Hyg. & Occup. Med.*, 4:482-493.

SPOLYAR, L. W.; KEPPLER, J. F., and PORTER, H. G. (1944): Cadmium poisoning in industry. *J. Ind. Hyg. & Tox.*, 26:232-240.

STEINBERG, H. H.; MASSARI, S. C.; MINER, A. C., and RINK, R. (1942): Industrial exposure to tellurium. *J. Ind. Hyg. Toxicol.*, 24:183-192.

STERNER, J. H. (1943): Determining margins of safety. *Ind. Med.*, 12:514-518.

STERNER, J. H.; OGLESBY, F. L., and ANDERSON, B. (1947): Quinone vapors and their harmful effects. *J. Ind. Hyg. & Tox.*, 29:60-84.

STERNER, J. H.; CROUCH, H. C.; BROCKMYRE, H. F., and CUSACK, M. (1949): A ten year study of butyl alcohol exposure. *AIHA Quart.*, 10:53-59.

STERNER, J. H. (1955): The experimental animal—man—in industrial hygiene. *AIHA Quart.*, 16:103-107.

STOKINGER, H. E. (1949): Toxicity following inhalation of fluorine and hydrogen fluoride. (In C. Voegtlin and Hodge, Pharmacology and Toxicology of Uranium Compounds). National Nuclear Energy Series, Div. VI, Vol. I, pp. 1021-56. McGraw-Hill, New York.

SUNDERMAN, F. W.; WEIDMAN, F. D., and BATSON, O. V. (1945): Studies of the effects of ammonium picrate on man and certain experimental animals. *J. Ind. Hyg. & Tox.*, 27:241-248.

SUNDERMAN, F. W., and KINCAID, J. F. (1954): Nickel poisoning. *J.A.M.A.*, 155:889-894.

SVIRBELY, J. L.; HIGHMAN, B.; ALFORD, W. C., and VON OETTINGEN, W. F. (1947): The toxicity and narcotic action of monochloromonobromomethane. *J. Ind. Hyg. & Tox.*, 29:382-389.

SVIRBELY, J. L. (1954a): Acute toxicity studies of decaborane and pentaborane by inhalation. *Arch. Ind. Hyg. & Occup. Med.*, 10:298-304.

SVIRBELY, J. L. (1954b): Subacute toxicity of decaborane and pentaborane vapors. *Arch. Ind. Hyg. & Occup. Med.*, 10:305-311.

SWENSSON, A.; HOLMQUIST, C. E., and LUNDGREN, K. D. (1955): Injury to the respiratory tract by isocyanates used in making lacquers. *Brit. J. Ind. Med.*, 12:50-53.

TABERSHAW, I. R.; FAHY, J. P., and SKINNER, J. B. (1944): Industrial exposure to butanol. *J. Ind. Hyg. & Tox.*, 26:328-330.

TOUSEY, R. G. (1954): Malathion—Cyanamid's versatile insecticide. *Agr. Chem.*, 9:No. 7, 49-50.

TREON, J. F.; CRUTCHFIELD, W. E., and KITZMILLER, K. V. (1943): The physiological response of animals to cyclohexane, methylcyclohexane and certain derivatives of these compounds. *J. Ind. Hyg. & Tox.*, 25:323-347.

TREON, J. F.; SIGMON, H.; WRIGHT, H., and KITZMILLER, K. V. (1949): The toxicity of methyl and ethyl acrylate. *J. Ind. Hyg. & Tox.*, 31:317-26.

TREON, J. F., and CLEVELAND, F. P. (1955): Toxicity of certain chlorinated hydrocarbon insecticides for laboratory animals, with special reference to aldrin and dieldrin. *J. Agr. Food Chem.*, 3:402-408.

TREON, J. F.; CLEVELAND, F. P.; CAPPEL, J., and ATCHLEY, R. W. (1956): The toxicity of the vapor of Arochlor 1242 and of Arochlor 1254. Paper read April 26, 1956 before the American Industrial Hygiene Association, Philadelphia.

U.S. DEPT. LABOR (1941): Control of welding hazards in defense industries. Division Labor Standards, *Spec. Bull. No. 5.*

U.S.P. H.S. (1943): Manual of industrial hygiene, W. M. Gafafer, editor. Saunders, Philadelphia, p. 264.

VIGLIANI, E. C., and ZURLO, N. (1955): Experiences of the Clinica del Lavoro with maximum allowable concentrations of industrial poisons. *Arch. Gewerbepath. u. Gewerbehyg.*, 13:528-535. (Abstracted in *Arch. Ind. Health*, 13:403, 1956.)

VON OETTINGEN, W. F.; HUEPER, W. C.; DEICHMANN-GRUEBLER, W., and WILEY, F. H. (1936): 2-Chlorobutadiene: Its toxicity and pathology and mechanism of action. *J. Ind. Hyg. & Tox.*, 18:240-270.

VON OETTINGEN, W. F. (1940): Toxicity and potential dangers of aliphatic and aromatic hydrocarbons. U. S. Public Health Service, *Pub. Health Bull. 255.*

VON OETTINGEN, W. F. (1941): The aromatic amino and nitro compounds, their toxicity and potential dangers. U. S. Public Health Service, *Pub. Health Bull. 271.*

VON OETTINGEN, W. F.; NEAL, P. A., and DONAHUE, D. D. (1942): The toxicity and potential dangers of toluene. *J.A.M.A.*, 118:579-584.

VON OETTINGEN, W. F. (1943): The aliphatic alcohols: their toxicity and potential dangers in relation to their chemical constitution and their fate in metabolism. U. S. Public Health Service, *Pub. Health Bull. 281.*

VON OETTINGEN, W. F.; DONAHUE, D. D.; SNYDER, R. K.; HORECKER, B. L.; MONACO, A. R.; LAWTON, A. H.; SWEENEY, T. R.; NEAL, P. A. (1944): Experimental studies on the toxicity and potential dangers of trinitrotoluene. U. S. Public Health Service, *Pub. Health Bull. 285.*

WAITE, C. P., and YANT, W. P. (1928): Microscopic pathology attending exposure of guinea pigs to vapors of ethyl bromide. U. S. Public Health Service, *Pub. Health Rpts.*, 43:2276-2282.

WAITE, C. P.; PATTY, F. A., and YANT, W. P. (1930): Acute response of guinea pigs to vapors of some new commercial organic compounds. IV. Ethylene oxide. U. S. Public Health Service, *Pub. Health Rpts.*, 45:1832-1843.

WATROUS, R. M. (1942): Methyl bromide, local and mild systemic toxic effects. *Ind. Med.*, 11:575-579.

WATROUS, R. M., and MCCAUGHEY, M. B. (1945): Occupational exposure to arsenic. *Ind. Med.*, 14:639-646.

WEAVER, F. L., JR.; HOUGH, A. R.; HIGHMAN, B., and FAIRHALL, L. T. (1951): The toxicity of methylal. *Brit. J. Med.*, 8:279-283.

WEBSTER, S. H. (1946): Volatile hydrides of toxicological importance. *J. Ind. Hyg. & Tox.*, 28:167-182.

WERNER, H. W.; MITCHELL, J. L.; MILLER, J. W., and VON OETTINGEN, W. F. (1943a): The acute toxicity of vapors of several monoalkyl ethers of ethylene glycol. *J. Ind. Hyg. & Tox.*, 25:157-163.

WERNER, H. W.; MITCHELL, J. L.; MILLER, J. W., and VON OETTINGEN, W. F. (1943b): Effects of repeated ex-

posure of dogs to monoalkyl ethylene glycol ether vapors. *J. Ind. Hyg. & Tox.*, 25:409-414.

WERNER, H. W.; NAWROCKI, C. Z.; MITCHELL, J. L.; *MILLER, J. W., and VON OETTINGEN, W. F. (1943)*: Effects of repeated exposures of rats to vapors of monoalkyl ethylene glycol ethers. *J. Ind. Hyg. & Tox.*, 25:374-379.

WILEY, F. H.; HUEPER, W. C., and VON OETTINGEN, W. F. (1936): On the toxic effects of low concentration of carbon disulfide. *J. Ind. Hyg. & Tox.*, 18:733-740.

WILSON, R. H., and DEEDS, F. (1936): Chronic nicotine toxicity. *J. Ind. Hyg. & Tox.*, 18:553-564.

WILSON, R. H. (1944): Health hazards encountered in manufacture of synthetic rubber. *J.A.M.A.*, 124:701-703.

WINSLOW, C-E. A. (1927): Summary of the National Safety Council study of benzene poisoning. *J. Ind. Hyg.*, 9:61-74.

YANT, W. P.; SCHRENK, H. H.; PATTY, F. A., and SAYERS, R. R. (1930): Acrolein as a warning agent for detecting leakage of methyl chloride from refrigerators. *U. S. Bur. Mines Rpts., Investigations 3027.*

YANT, W. P.; SCHRENK, H. H.; WAITE, C. P., and PATTY, F. A. (1930): Acute response of guinea pigs to vapors of some new commercial organic compounds. II—Ethyl benzene. U. S Public Health Service, *Pub. Health Rpts.*, 45:1241-1250.

YANT, W. P.; SCHRENK, H. H.; and PATTY, F. A. (1932): The toxicity of dichlorotetrafluorethane. U. S. Bur. Mines Rpts., Investigations 3185.

YANT, W. P.; PATTY F. A., and SCHRENK, H. H. (1936): Acute response of guinea pigs to vapors of some new commercial organic compounds. IX—Pentanone (methyl propyl ketone). U. S. Public Health Service, *Pub. Health Rpts.*, 51:392-399.

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Dr. Henry Field Smyth, Jr., receiving the Cummings Memorial Award from N. V. Hendricks, President of the American Industrial Hygiene Association. The presentation was made at the banquet of the Association's Seventeenth Annual meeting in Philadelphia, Wednesday, April 25.